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APHASIA IN CHILDREN

by

ALEX. W. G. EWING, M.A., Ph.D.,

With an Introduction by

E. D. ADRIAN, M.D., F.R.C.P., F.R.S.

Fellow and Lecturer of Trinity College, Cambridge.

Contents include:—Nature of the Problem—Hearing Tests—Value of the Audiometer—Hearing Capacity of Normal, Aphasic, and Deaf Children—Hearing Capacity and Speech Development—The Literature of Aphasia—The Speech and Language of Aphasic Patients—Aural Examination of High-Frequency Deaf Patients—Summary of Conclusions—Tables of Experimental Results—Bibliography.

The aphasic child is necessarily a source of deep distress to its parents, and commences life with a grave personal handicap. Methods of treatment have not generally met with success, and a contributory cause of failure has undoubtedly been our limited understanding of the etiological factors. The work of Dr. Ewing has disclosed the existence of a peculiar form of deafness which in his view provides a key to the problem. Of even greater importance is his conclusion, founded upon the results of a series of carefully conducted experiments, that in certain cases at least it is possible, by relieving the aural defect, to remedy the aphasia. His book gives a clear account of his methods of investigation, and should certainly be read by all likely to be concerned with speech defects in children.

pp. 165. 9 *Tables*, 2 *Charts*. 10/6 *net*.

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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. XI. JULY, 1930. No. 41.

Original Papers.

LANDRY'S PARALYSIS: A CLINICAL AND PATHOLOGICAL STUDY.

By

F. GOLDBY, London.

INTRODUCTION.

In 1859 Landry described the first case of a kind of acute or subacute paralysis which since then has become associated with his name. In their clinical features these cases are sufficiently dramatic to attract attention, and in their incidence sufficiently rare to merit individual publication, so that the subsequent literature has been plentifully supplied with reports of cases and discussions concerning them. Landry himself described a clinical syndrome which was fairly definite, but which was associated in the cases described by him with a remarkable lack of post-mortem findings. With more modern technique for examining the nervous system it is likely that changes would have been found which he failed to find, but even by these methods it is probable that they would be remarkably slight. Without entering upon a discussion of the nature of disease, it may be said that diseases have traditionally been described as entities on the basis of a common etiology, a common syndrome or complex of symptoms and physical signs, and a common pathology. With Landry's paralysis little or nothing is known of a common etiology, and the pathological findings, in so far as they are not negative, have been variable; consequently a diagnosis of 'Landry's paralysis' must rest upon the most unsatisfactory and variable member of the triad, viz., upon a complex of symptoms and physical signs. The essentials of this complex, in its typical form, are an acute or subacute paralysis of lower motor neurone type ascending from the feet upwards, symmetrically on both sides of the body. There is no objective sensory disturbance, and no loss of function of the sphincters of bladder or rectum. There is little or no febrile reaction, and no considerable muscular atrophy. The common result is death from paralysis of the motor nuclei of the bulb. It is proposed in this paper to describe three hitherto unreported cases which corresponded in their clinical features more or less closely with this picture, together with the post-mortem findings in two of them; further,

to compare them with the reports of 52 cases collected from the literature, in the hope of answering the question whether there is such a disease as Landry's paralysis with a uniform etiology, symptomatology and pathology, or, if not, what the relation of this particular symptom-complex is to etiological and pathological factors, and how far it can be related to other and more strictly defined diseases.

Before passing to an actual description of cases, some mention must be made of the criteria which have been used in judging whether a case should be included in this series or not. Practically any case which has been reported under the name of Landry's paralysis or acute ascending paralysis and which shows no evidence of involvement of the upper motor neurone, and no, or very slight, objective sensory disturbance has been included, provided any atrophy of muscles which occurs is insignificant compared with the paralysis. With a few notable exceptions all the cases have been collected from English or American literature on account of its greater accessibility.

I have to thank Dr. S. A. Kinnier Wilson for permission to make use of the first case, W. A., and Dr. D. Firth for the other two, R. T. D. and J. L. W.

PERSONAL CASES.

Case 1. W. A., age 50, male. Admitted to King's College Hospital, April 8, 1928, under the care of Dr. Kinnier Wilson.

A spare man, with rather poorly developed musculature, of temperate habits. No significant past history of disease. He had been for a fairly long walk on the day before the onset of symptoms.

He first noticed a dull aching pain in his left foot, which extended to the calf and, on the second day, up to the level of the navel and thence to the right arm. On the third day pain of a similar character was felt in the right foot, and he found that his legs gave way under him. Later in the same day his left arm became useless and his right arm weak. He remained in much the same condition till towards evening on the fourth day, when he had great difficulty in breathing and was particularly troubled by some phlegm in his throat which he was unable to cough up. He was admitted to hospital that same evening.

His respirations were shallow, and at the rate of about 40 to the minute; the pulse was 140, regular, and of poor volume and tension, the radial artery being tortuous and easily palpable. Temperature 99° F. Examination of the lungs revealed very poor air entry on the right side, but no adventitious sounds or impairment of note. Breathing was diaphragmatic. The area of cardiac dulness was almost completely obscured by emphysema; the sounds were normal but distant. B.P. 120/90. The abdomen was lax and slightly distended, but not otherwise abnormal. The urine was normal, and there was no disturbance of function of the sphincters of the bladder or rectum.

With the exception of the diaphragm there was almost complete flaccid paralysis from the pectoral muscles downwards. Slight flexion and extension of the toes was possible. Deep and superficial reflexes were all absent except that the plantar responses were both feebly flexor. The left arm was completely paralysed, with loss of deep reflexes; the right arm was very weak, but feeble movements of the fingers were possible. The

biceps jerk was just present, the triceps jerk very doubtful and the supinator jerk absent. All movements of the neck could be performed weakly; he could just raise his head from the bed. The cranial nerves showed no abnormality. The fundi were normal, and mastication, deglutition and articulation were all normally performed. No sensory changes could be detected.

On the fifth day there was no notable change. The temperature was, morning 98·4° F., evening 101·6° F., the pulse 140, and respiration 40. Air entry on the right side of the chest had improved a little.

Lumbar puncture was performed. The fluid issued under what appeared to be slightly increased pressure. It was clear and contained 4 lymphocytes per c.mm. Total protein ·06 per cent. (by Mestrezat's diaphanometric method); globulin test (Ross-Jones' modification of the Nonne-Apelt reaction) negative. Sugar and chlorides were both within normal limits, and the Wassermann reaction was negative. No organisms were found.

In the evening of that day (the fifth) it was found that he could move the terminal phalanx of his left thumb, which he could not do before, and he thought the movements of his right arm and toes were stronger. They were not obviously so. Coarse râles could be heard over the right lower lobe.

On the sixth day it appeared as if he could raise his head a little more easily from the bed, and slight flickering contractions of the pectoral and left biceps muscles could be obtained. There was still no objective sensory change, and no sphincter trouble.

The patient died that evening of broncho-pneumonia.

Post-mortem examination (No. 75, 1928, in hospital records).

Lungs: Severe broncho-pneumonia most marked at the right base. Some œdema of the apices with a tuberculous scar on the left. *Larynx* and *trachea* inflamed. *Heart:* Some petechial hæmorrhages over the left ventricle. Patchy thickening of pericardium over the right auricle. *Abdomen:* Intestines distended with gas. Liver: Slight fatty change. Spleen: Normal. Kidneys: Congested. *Spinal Cord:* All superficial veins engorged with blood. *Brain* apparently normal.

Unfortunately only the spinal cord with its membranes and nerve roots was preserved for sectioning.

The *membranes* were in all sections normal. Their blood-vessels were perhaps overfilled with blood but there was no evidence of inflammatory infiltration. The blood-vessels in the cord itself were also very full of blood, but again there was no evidence of inflammatory infiltration.

The *nerve tracts* in the cord and the spinal *nerve roots* had a normal appearance in sections stained with hæmatoxylin and eosin, but Marchi's method was not used.

The *nerve cells* were examined after staining with Nissl's methylene blue. About one-third of them were normal in appearance. The rest showed changes which varied from complete disappearance of granules, with excentricity of the nucleus and a rounding and blurring of cell outlines, to cells which showed only some diminution in size of the granules. This gave them a fine dust-like appearance, often only at one pole of the cell, or immediately round the nucleus. The extreme change was not very common, nor could it be said that such chromatolysis as there was, was predominantly perinuclear. In the lumbar and sacral parts of the cord the process was most advanced; in these parts it was difficult to find a completely normal nerve cell, whether in the anterior or posterior horns. In the thoracic region chromatolysis was still well-marked but normal cells were commoner. The cells of Clarke's column and those at the base of the posterior horns seemed more affected that those in the anterior horns themselves. In the cervical region changes were slight, and again most easily found in the intermediolateral parts of the grey matter.

The neuroglia showed no change, but special neuroglial stains were not used. The central canal of the cord was patent down to the sacral region.

Case II. R. T. D., age 25, male. Admitted to King's College Hospital, November 26, 1927, under the care of Dr. Firth. Nothing is known of the past history of this patient. He was a well-developed muscular young man.

His illness started with headache and pain in the back. He vomited once. The pain and headache continued and on about the tenth day his legs rapidly became paralysed. Two days later he was admitted to hospital, when a complete flaccid paralysis of the arms, lower intercostals, diaphragm, abdominal muscles, and legs had supervened. A little feeble movement of the toes was possible, and the plantar reflexes were flexor. Slight early papillœdema was noted in both fundi, but otherwise the cranial nerves were normal. Pain persisted in the back, and he vomited once after admission. There was no objective sensory change and no loss of sphincter control.

A lumbar puncture was performed. Unfortunately the report on the fluid obtained has been lost, and it is only known that a pleocytosis, mainly (? wholly) of lymphocytes was present.

He died about 12 hours after admission to hospital. His temperature during this time was in the neighbourhood of 101° F.

Post-mortem examination (No. 239, 1927, in hospital records).

Lungs: Collapse of both lower and the posterior parts of both upper lobes, more complete on the right than on the left. Tuberculous scars at both apices. *Heart:* Right side engorged. *Aorta:* Slight early atheroma. *Intestines:* Distended with gas. *Brain:* Basal ganglia rather pink in colour. Otherwise normal. *Cord:* The veins on the posterior aspect were engorged, and the cord itself very soft and slightly pink. The other organs were all normal.

Microscopic sections showed the following pathological changes.

The *meninges* of the cord and round the brainstem showed slight general infiltration chiefly with lymphocytes, but also with a few plasma-cells and an occasional polymorph. This infiltration was much more marked in the immediate neighbourhood of the blood-vessels, which were engorged with blood. It was most conspicuous in the lumbar region, less so round the brainstem, and least of all in the thoracic region. Perivascular infiltration with small round cells was also very marked round the *blood-vessels of the cord* and brainstem. It was most conspicuous round the medium-sized vessels, the Virchow-Robin spaces being packed with lymphocytes, but, particularly in the lumbar region, it extended to the small vessels and capillaries. All the vessels were engorged, but there was no evidence of thrombosis or hæmorrhage; nor was there any evidence of inflammatory infiltration in the nerve-roots.

The *nerve-cells.* In the lumbar region Nissl's stain showed an almost complete chromatolysis of all the nerve-cells, both in the anterior and in the posterior horns. A few cells still preserved a few granules in the peripheral parts of their cytoplasm. Many were swollen and rounded, with nuclei excentric and badly stained. In the thoracic region these changes were even more advanced. Most of the cells were rounded and had lost their processes; some were vacuolated, and many showed excentricity and poor staining of the nuclei. Only a very few retained any Nissl's granules. The changes were again general, but more marked in the anterior horns. The section through the brainstem was made at just below the upper limit of the gracile and cuneate nuclei. The nerve cells of the hypoglossal nucleus were practically normal in appearance, as were those of the eleventh nerve nucleus, although the latter did show some signs of perinuclear chromatolysis. The cells of the gracile and cuneate nuclei and of the olive showed rather more definite perinuclear chromatolysis, and those granules which were present seemed smaller and less definite than usual. On the whole, however, degenerative changes in the cells in this region were slight, and nowhere so marked as in the cord.

There was some *neuroglial* proliferation in the cord, especially in the anterior horns of the thoracic part. The central canal was patent in all the sections examined,

Sections of the motor cortex and of the ulnar, posterior tibial and external popliteal nerves, stained with hæmatoxylin and eosin, showed no abnormality. Marchi's method was not used.

It is noteworthy that in the thoracic part of the cord, where the degeneration of nerve cells and neuroglial proliferation were most marked, there was least inflammatory infiltration. It was confined almost exclusively to the sheaths of the vessels dipping into the cord, and to their larger branches.

CASE III. J. L. W., age 16, male. A case seen privately by Dr. Firth, to whom I am indebted for the following details.

Past history. Whooping cough, catarrh of the stomach, an abscess in the neck, and measles, all before the age of ten years.

On November 5, 1927, the patient complained of headache and general malaise.

On the second day of his illness he still felt vaguely unwell, but a little better, and on the third day went to work as usual. He complained of nothing further till the sixth day when headache became more intense, and he took to his bed. He was very restless. On the seventh day he was unable to hold his head up and complained of pain in his neck. This pain moved downwards later between his shoulders and to the level of his waist. He was sleepless, but was still able to get out of bed. On the eighth day he was worse, but got up. There were pains in the back of the neck, shoulders, knees and ankles. He had to go back to bed. His head was retracted and he was unable to raise it from the pillow. He vomited once, and had some difficulty in swallowing. The temperature was 103° F.

On the ninth day his temperature was still 103° F. He vomited several times, and the difficulty in swallowing increased. His left arm became completely paralysed. During the night he was delirious, and his temperature rose to 104° F. On the tenth day he was seen by Dr. Firth. He was lying in bed with an anxious expression, breathing rapidly and feebly. The cranial nerves were normal, and articulation was normal. There was complete paralysis of the left arm, lower intercostal muscles, diaphragm and abdominal muscles, and of the legs. Slight movement of the right hand and forearm was possible. No deep reflexes could be obtained. There was no objective sensory disturbance. He died on the evening of the same day.

There is no mention of sphincter trouble, or of any other physical sign in either chest or abdomen.

A post-mortem examination was not allowed.

ANALYSIS OF CASES.

Fifty-four cases in all have been collected and tabulated. They all show a paralysis of the lower motor neurone type affecting both sides of the body and of wide distribution. None shows any considerable objective sensory disturbance, and only two of the recovered cases wasting that was at all marked. In none was there any clinical evidence of pyramidal involvement. The mortality of the whole series is high—70 per cent. These features alone are enough to separate it fairly definitely from typical cases of anterior poliomyelitis, polyneuritis, myelitis, or the described muscular atrophies. It is obvious, however, that it is not sufficiently homogeneous for a detailed analysis of clinical and pathological findings to be of value without subdivision into groups, but one or two general observations may be of use.

The series, as remarked, shows a *mortality* of 70 per cent. *Males* constitute 80 per cent. of the cases. The *age-incidence* is as follows :

Age in years	0—10	10—20	20—30	30—40	40—50	50—60	60—70	70—80
No. of cases	3	10	23	8	3	4	3	0

The youngest was two and a half years, and the oldest 64, so that, though practically no age is exempt, by far the heaviest incidence is during young adult life, chiefly in the third decade. With regard to *occupation* it can be said only that men of the labouring class, and those most exposed to cold and poor conditions generally seem to be affected chiefly, and town dwellers more than countrymen. This is more an impression gained while reading the literature than a conclusion drawn from a comparison of records, which were too often incomplete for anything more to be possible.

In reviewing the series it became clear that several of the cases were too incompletely reported to be of value in a detailed analysis. For this reason 13 were rejected, and the following analysis has been made on the remaining 41. Even these were often not described in the detail one could have wished, so that for some of them classification has of necessity been speculative, and any conclusions drawn from it cannot be more than provisional.

A preliminary grouping was made as follows :

I. Cases with post-mortem findings.
   Further divided into :
   (*a*) Those without evidence of inflammatory infiltration of the nervous system.
   (*b*) Those with inflammatory infiltration of the nervous system.
II. Fatal cases reported without post-mortem findings.
III. Cases which recovered.

Each group was analysed separately and it was found that in each (including the two subdivisions of Group I) three roughly distinct types could be differentiated, and further that in each group these three types were approximately the same. To give the analysis of each of these groups in full and separately would obviously involve a great deal of needless repetition, so that the plan adopted here has been to analyse the series as a whole, using the three types mentioned as a basis. Before proceeding, however, something must be said about the constitution of the three types. As will be seen little help can be obtained from considerations of etiology since too little is known of it ; subdivision will have to depend on clinical and post-mortem findings. These findings are no more or less than the evidence that can be found of damage to the organism, and reaction on the part of the organism, not only to the

noxious agent directly, but to the presence of tissue already damaged by that agent. Such a classification therefore will simply be a classification of types of injury and reaction, none of which is necessarily specific for any single noxious agent. It has been found further, that no one factor, whether clinical or pathological, could be used to differentiate any two types absolutely, without, as judged by other factors which one has no reason to look upon as less important, making obviously forced and artificial subdivisions. This point was brought out particularly clearly when the attempt was made to classify on the basis of the presence or absence of inflammatory infiltration. Both inflammatory and non-inflammatory groups fell roughly into three types, six in all. Calling these for the moment Types a, b, c, and Types d, e, f, it became clear that e, for instance, might have more in common with, say, a, than with d or f, and was more naturally grouped with it. It follows that a classification, to be of any value, must be based on as many factors as possible taken together; in other words that it must be a classification of groups of factors, although it may be justifiable at times to lay more stress on one factor than on another. The result of applying this method here has been to reduce the series to three groups, in which the following distinguishing factors seem to be of preponderating importance.

I. An acute ascending paralysis with little or no febrile reaction and a remarkable slightness of post-mortem findings.

II. An acute ascending paralysis not uncommonly associated with sensory changes suggestive of peripheral neuritis, and with the signs of general toxæmia but little febrile reaction; with post-mortem findings, which, though variable, generally show both parenchymatous and interstitial changes in the central nervous system and peripheral nerves.

III. An acute ascending paralysis preceded or accompanied by an acute febrile illness and with post-mortem findings resembling those of anterior poliomyelitis.

How far this grouping is justifiable will be seen from the detailed analysis of the 41 cases which follows. The cases have been tabulated as shortly as possible. It was difficult in many of them to estimate the duration accurately, since the onset was often insidious. The rule followed has been to date the illness from the first day when any paralysis or weakness was noted. This, of course, is purely arbitrary. The paralysis, except where it showed abnormal features, has not been described in detail; where not otherwise stated it may be taken that it was of the flaccid ascending type, and approximately symmetrical.

I. There are 11 cases in this group four of which recovered, giving a mortality of 60 per cent. There was only one female, so that roughly 90 per cent. are males.

Age distribution lay between 10 and 60 years, with some preponderance between the ages of 20 and 30. The average age of the whole group was 30

years ; of the fatal cases alone, 33 years, and of the non-fatal alone, 25 years.

The duration of the illness in fatal cases varied between 4 and 40 days, with an average of 15 days. The paralysis was in all cases of the ascending type except in one, where it began in the arm. Bulbar involvement was mentioned in 75 per cent. of the cases, usually before the twelfth day. Wasting, or change in the electrical responses, was not reported in any case, although the latter point was investigated in only a few of them. In 3 cases (25 per cent.) paralysis seemed rather less complete in the distal than in the proximal segments of the limbs. This may well be due to the greater mechanical advantage at which the distal muscles act. Slight sphincter disturbance occurred in two cases, where it was almost certainly due to profound general weakness. In one, however, it is stated that the bladder was paralysed before the skeletal muscles.

Sensory changes.—Subjective : Paræsthesiæ, pains, numbness, heaviness, or aching were reported in 75 per cent. of cases. Objective : Very slight changes in three. These consisted in slight blunting of sensation on the soles or legs, or slight tenderness of calves and arms. It would seem quite justifiable to say that for all practical purposes there is no objective sensory change reported in this group. Febrile reaction was negligible in all cases but two : in one case it was undoubtedly due to a terminal broncho-pneumonia, and in another it is of doubtful significance. This case was atypical also in the early bladder paralysis, and one had some hesitation in including it in this group.

The cerebrospinal fluid was examined in only one case, and was then found to be normal.

Pathology.—Chromatolysis, more or less advanced, was found in the nerve cells of the cord in all cases. The three cases reported by Buzzard (1907) showed some diffuse fatty change in the peripheral nerves and in the nerve fibres of the central nervous system. He himself points out that this change is not specific and may be found as a result of any profound toxæmia, and that it is not associated with any loss of function. It is probable that similar changes would have been found in the other cases had they been looked for carefully enough.

An enlarged spleen was mentioned twice, both times in association with tubercle ; in one, old pulmonary tubercle, and in the other, with an area of tuberculous meningitis in the mid-dorsal region.

Except for the possibility of tubercle in the two above mentioned cases, and a very speculative connection with syphilis in two others and with wetting in one more, no etiological factors can be suggested for any of these cases.

In the non-fatal cases recovery was apparently complete, the whole duration of the illness varying between approximately six weeks and one year.

II. This group contains 20 cases of which 9 recovered, giving a mortality of about 50 per cent. The age varied between about 5 and 60 years with an average over all of 28 years. The fatal cases alone gave an average of 30 years the others an average of 26. Women constituted about 25 per cent.

of the cases. In the fatal cases the duration varied between 4 and 42 days, with an average of 15 days.

It is in this group that differences from the classical Landry type were most marked, although the paralysis was ascending in all but one case.

Bulbar involvement was mentioned in about 75 per cent. of cases; a conspicuous feature was the occurrence of ophthalmoplegias with or without paralysis of one or both seventh nerves, particularly in the non-fatal cases. Ophthalmoplegia, as evidenced by squint, diplopia or ptosis, was noted in six (25 per cent.), and weakness of one or both seventh nerves in about the same proportion. Ophthalmoplegia and facial weakness were each mentioned only once among the 11 fatal cases.

Changed electrical reactions were mentioned five times. In one case the muscles reacted badly to faradism, and in another the nerves were less excitable than the muscles to faradism. In a third, a definite reaction of degeneration developed five weeks from the onset; in a fourth, there was diminished response, but no reaction of degeneration, and in a fifth, reaction of degeneration in the anterior tibial muscles. As would be expected, these changes seem rather more definite among the recovered than among the fatal cases.

Wasting of muscles was mentioned in four cases (20 per cent.). In one fatal case of rather long duration the wasting was most marked in the shoulder muscles; it was slight and general in two others, and in a fourth confined to the extensors of the wrists, the small muscles of the hands and the anterior tibial muscles, but came on a considerable time after the onset of the illness.

Sphincter disturbance was mentioned in five cases (25 per cent.). In all but one (where there was definite double incontinence) it was slight in degree and probably unimportant.

Sensation.—Subjective disturbance was mentioned in all but three cases, i.e., in more than 80 per cent. These consisted of pains, paræsthesiæ, and a feeling of numbness or heaviness in the limbs. Aching and backache were not commonly mentioned. Objective changes of a minor degree were common, being mentioned in nearly 50 per cent. In three cases this consisted in tenderness over nerve trunks, in four, muscle tenderness; hyperæsthesia was mentioned twice and impairment of sensation on arms and thighs, and indefinite impairment on the legs, once each.

Febrile reaction and toxæmia.—Febrile reaction was mentioned in only two cases, in one of which it was probably due to coexisting bronchitis. In 11 cases (about 50 per cent.), there was more or less marked general toxæmia. This varied from simple constipation to constipation with colic and definite hæmatoporphyrinuria. In other cases the evidence was very varied; vomiting and headache with septic teeth; fever following vaccination; delirium, labial herpes, stomatitis and coryza; Grave's disease. Reddish or claret-coloured urine was mentioned in three cases; plumbism and chronic nephritis in one. One may speculate that in those cases where the urine was noted to

be reddish or claret-coloured the condition has been hæmatoporphyrinuria, the more so that in one of them there was a history that trional and sulphonal had been taken.

Examinations of blood and cerebrospinal fluid were too few to justify any general conclusions.

Pathology.—The changes found in the nervous system were very variable, but in all cases both spinal cord and peripheral nerves were affected. They varied from chromatolysis of the nerve-cells of the cord combined with more or less parenchymatous degeneration in the peripheral nerves, to advanced degenerative changes in the nerve-cells of the cord, with degeneration in the tracts and perivascular round-celled infiltration, and, in the peripheral nerves, marked parenchymatous and interstitial change. The latter consisted in swelling and fragmentation of myelin, swelling of the axis-cylinder, proliferation of the cells of Schwann and inflammatory infiltration. Simple interstitial neuritis was mentioned in one case. In another, changes in the peripheral nerves were very marked, while in the cord the tracts were normal and the nerve-cells showed only a low degree of chromatolysis.

There was nothing notable in the other organs, except that the spleen was enlarged in two cases.

Etiology.—Clearly any of the toxæmic conditions already mentioned may have been factors in the etiology. In addition there were three cases in which syphilis was possibly a factor, in one combined with alcohol. Alcohol and exposure in one, and exposure alone in another complete the list of observed etiological factors for this group.

Recovery.—This was practically complete in five cases. In one of them the pupil light reflex had not returned after eight weeks, and on this account the possibility of syphilis was considered. The whole duration in these cases varied from six to eleven weeks. In four others there were more or less definite sequelæ ; in one the knee jerks were still absent after two years ; in another, reaction of degeneration appeared after five weeks, but the further progress of the case is not reported. In a third there was some general wasting, and, in a fourth, wrist and foot drop. These two cases were followed up for nine weeks and 13 months respectively.

III. The third group contains 10 cases ; only two of these, unfortunately, were reported with post-mortem findings, so that, for the most part, they have been grouped together on clinical grounds. There were only two recoveries, giving a mortality of 80 per cent. The average age was 18 years. No patient was younger than 10, and none older than 30 years. There were no females. The duration of the fatal cases varied between three and 12 days, with an average of seven.

Paralysis.—This was fairly typically ascending except in three cases (33 per cent.). In two the arm was first affected and in the third the shoulders and neck. Bulbar involvement was mentioned in 50 per cent. There was

no report of the electrical reactions. Wasting was mentioned in only one case. Here the legs were markedly wasted and the knee jerks lost after 20 days. *Sphincter disturbance* was relatively frequent. It was reported in 60 per cent. Usually slight in degree, there was nevertheless complete double incontinence in one instance. It is probable that in these cases incontinence is due rather to the severe constitutional illness than to actual nervous lesions of the centres or tracts controlling the bladder and rectum.

Sensation.—Subjective changes such as paræsthesiæ, numbness, etc., were remarkable from their rarity. On the other hand such symptoms as backache, headache, and general malaise were common. Apart from slight hyperæsthesia, no objective changes were mentioned.

Febrile reaction.—Fever was marked in all cases but two, in which it did not rise above 100° F. The first of these was an unusual case in that the condition was apparently associated with *B. anthracis*. The fever was very commonly associated with headache, vomiting, and other signs of an acute febrile illness, and in three cases with stiff neck or head retraction.

The cerebrospinal fluid in one case showed a pleocytosis.

Pathology.—As was said above, there are only two post-mortem reports in this series. One of these (Case 2 above) has already been described among my personal cases. The other showed hæmorrhagic softening of the anterior horns with marked degeneration of nerve cells and perivascular infiltration. Many organisms resembling *B. anthracis* were present.

Etiology.—Apart from the anthrax mentioned above, severe wetting and chill in one case, and previous recent vaccination in another, no possible factors could be suggested.

Recovery.—In the two non-fatal cases, wasting of the legs and absent knee-jerks were noted in one, which was followed up for only 20 days, and in the other, weakness of the left leg, persisting six months later.

It will be well at this point to recapitulate as shortly as possible the main points which have been brought out in this analysis, laying especial stress on those which serve to distinguish the different groups one from another.

I. This is a form of acute ascending paralysis, having its incidence almost exclusively on males and usually during the third decade of life. The case mortality is high, 60 per cent., but the prognosis appears to be slightly better under the age of 30 than above it. The paralysis is almost, but not quite always ascending in type; it is not associated with wasting, or, apparently, with changed electrical reactions. This last point needs more careful investigation. Slight sphincter disturbance is not uncommon, and is probably due to general weakness. Bulbar involvement is usual, but the nuclei of the third, fourth, fifth, sixth and seventh cranial nerves are not as a rule affected. Sensory changes are almost entirely subjective, and they take the form of paræsthesiæ, pains, aching, numbness, etc., usually in the limbs and often preceding the onset of paralysis by a few days. They are present in nearly

all cases. Febrile reaction is conspicuous by its absence, nor are there any signs of meningeal irritation or of increased intracranial tension. The cerebrospinal fluid in one case was normal.

Post-mortem findings are limited to vascular engorgement of the meninges, chromatolysis of the nerve cells of the cord, and slight diffuse fatty change in nerve tracts and peripheral nerves, not of a specific nature. Recovery, when it occurs, is complete, but may take a year or more. When fatal, the whole duration is commonly about 15 days, and death usually results from respiratory paralysis or from bronchopneumonia. No points of prognostic significance can be suggested.

Nothing can be suggested with regard to etiology.

II. This group differs from Group I in that it has a smaller mortality, 50 per cent., and that the incidence is higher in females, although once more males predominate. The age incidence is not markedly different, and again the prognosis seems slightly better under than over the age of 30. Again the paralysis is almost always ascending in type. It differs from the paralysis in Group I in that motor cranial nerves are commonly involved, usually asymmetrically, and also in that changed electrical reactions and wasting are sometimes observed, although only in a minority of cases. Sphincter disturbance is rather more common, but generally of the same type. Sensory changes of a subjective nature do not differ appreciably from those in Group I. Objective sensory changes, often of the nature of tenderness over nerve trunks occur in half the cases, but are always slight ; they are sufficiently definite, however, to constitute a point of distinction. Signs of a general toxæmia are common, so that, in spite of the absence of febrile reaction, there is more constitutional illness than in Group I.

Post-mortem changes in both cord and peripheral nerves are always present and of a parenchymatous nature, with or without inflammatory reaction. Recovery is often complete in two or three months ; sequelæ, such as wasting and absent deep reflexes, are not uncommon. Fatal cases last usually about 15 days, and again no point of prognostic importance transpires.

Etiologically there seems to be some correlation with factors which are known to be of importance in causing a polyneuritis.

III. In this group the mortality is high, 80 per cent., and the age incidence definitely low—as a rule under 20 years. The preponderance of males is very marked. Paralysis is not always so typically ascending, and shows a tendency to start first in one limb and to become generalised later. Bulbar involvement is common, and is more like that of Group I than of Group II. Wasting is mentioned in one of the recovered cases ; clearly most of these cases were of too short duration for wasting to have occurred. Sphincter disturbance is more frequent than in either of the other groups, and this is probably associated with the acute nature of the illness. The most characteristic feature of this group is the marked febrile reaction, often associated with signs of increased intra-

cranial tension and meningeal irritation. Sensory changes, both objective and subjective, are almost completely absent. The cerebrospinal fluid in one case showed a pleocytosis.

Post-mortem the changes are found to be strikingly like those of anterior poliomyelitis. Only two cases recovered, but in both of these recovery was not complete. There was wasting of the legs in one, and weakness of one leg in the other. The fatal cases have a duration on an average of only about half that in either of the other two groups.

The etiology remains obscure.

DISCUSSION.

It is fairly clear from this description of cases of acute ascending paralysis that it is the cases in Group I which correspond most closely with Landry's classical description. Before discussing them further the position of Groups II and III must be made clear, since they seem to have a more obvious connection with better known morbid conditions.

Group II appears to be fairly closely related to cases of multiple neuritis. Such cases form a very heterogeneous group. They have no single etiology but are related to a variety of toxic conditions, the toxin being sometimes exogenous, sometimes endogenous or metabolic, and sometimes the result of an infection, like diphtheria, or of an acute fever of unknown causation. In addition, there are a certain number of cases for which no known toxin can be suggested, or where the absorption of an unknown toxin from the gut is postulated. So far they correspond fairly closely with the cases in Group II, many of which showed evidence of toxæmia. It is significant that the group contained one case of acute hæmatoporphyrinuria, and three others in which hæmatoporphyrinuria was a possibility. It is said (Löffler, 1919), that of 14 recorded cases of acute hæmatoporphyrinuria no less than five have shown the syndrome of Landry's paralysis. Further, it is well known that hæmatoporphyrinuria, whether of the paroxysmal idiopathic variety, or following the administration of sulphonal, trional, etc., is commonly associated with multiple neuritis, and, as in other forms of multiple neuritis, this may affect either the sensory or motor nerves predominantly. According to Harris (1926) retention and even incontinence may occur, and also mental symptoms like nervousness and excitability and even epileptiform fits. Gastro-intestinal symptoms, such as colic, constipation and vomiting are almost the rule.

The character of the paralysis in Group II differs from that which usually occurs in clear cases of multiple neuritis; it is on the whole more generalised, and usually ascending. Similarly sensory changes, although present in many cases, are minimal. There are not sufficient grounds for setting them apart as a separate disease. Irregular and indefinite areas of anæsthesia and hyperæsthesia, and tenderness over the nerve trunks and muscles are sufficiently common and characteristic to suggest multiple neuritis very strongly. Moreover, it is a feature of cases of multiple neuritis that sometimes the sensory

nerves and sometimes the motor nerves are affected almost exclusively; diabetic neuritis is usually mainly sensory, and lead neuritis is often mainly motor, while the two extreme types are sometimes the result of the same toxin in different patients. It would seem that the cases in Group II represent simply the extremes of a series, and not a distinct condition. The same argument applies to the fact that the paralysis is more than usually extensive in the cases in Group II, so that only its ascending nature remains to distinguish it from more ordinary cases of multiple neuritis. The frequency of the involvement of the eye and facial muscles is noteworthy; although similar involvement is well known in cases of multiple neuritis, it does not occur so often. The electrical reactions were not reported often enough for any useful discussion to be made about them. Sphincter disturbance was not uncommon, but then again this may occur in cases of multiple neuritis. The post-mortem findings were variable, but so they are in cases of multiple neuritis; parenchymatous and interstitial changes are both found, sometimes separately, and sometimes together. When they occur together it is possible that the one may be the result of the other; what the sequence is, is very difficult to say, although at times one may be able to make a more or less probable guess. After all, one would not expect that, in a group of cases with so varied an etiology, the post-mortem changes would be uniform.

They differ from the usual cases of multiple neuritis in that the central nervous system is far more affected than would be accounted for by a pure peripheral neuritis. It is possible that in some of the cases the changes in the central nervous system were secondary to those in the peripheral nerves, but this cannot be maintained for more than a minority of them. Nevertheless lead, for example, is known to affect the motor neurone inside as well as outside the nervous system; permanent atrophy of the intrinsic muscles of the hand may result from it, and even, rarely, a generalised progressive muscular atrophy. In other cases of multiple neuritis too, permanent atrophies may result which are probably evidence of damage to anterior horn cells. Consequently, once again, there is no evidence that Group II is more than a collection of extreme cases selected from the large and heterogeneous group called multiple neuritis. The position seems the more secure in that several cases intermediate in character between these cases and the more typical cases of multiple neuritis can be found reported in the literature. They have usually been reported as cases of Landry's paralysis. For example, Stockton (quoted by Diller, 1902) describes the following case. A female, age 27, suffered with nervousness, insomnia and generalised pains for two weeks. The gluteal region was then noted to be anæsthetic and she was incontinent. Paralysis started in the legs and spread up to the arms, both arms and legs becoming anæsthetic at the same time. The patient had been given trional, and hæmatoporphyrinuria was present throughout her illness. This would appear to be a case of ascending polyneuritis which differs from those just described only in the extent of the anæsthesia. Diller (1902) reports the case of a man

aged 25, who, three weeks after he had developed a hard chancre, and while presenting typical signs of secondary syphilis, was seized with severe pains and general weakness and lassitude. Six weeks later he was admitted to hospital with much general emaciation, great weakness of his legs, and rather less of his arms. Knee-jerks, which at first were exaggerated, were soon lost. There were fibrillary twitchings in his muscles. One would have little hesitation in diagnosing this case as one of multiple neuritis due to syphilis, yet the neuritis is clearly very widespread and no mention is made of any objective sensory changes. Progressive muscular atrophy of syphilitic origin is a possible alternative diagnosis. An even clearer case is one reported by Green (1901). A boy aged 3 years and 10 months had an attack of diphtheria. Fourteen days later there was dysphagia, and paralysis of the soft palate and weakness of the legs. Later the pharyngeal muscles became paralysed and the left side of his neck weak; simultaneously the weakness of his legs increased, and finally spread up to involve the arms. He died of respiratory paralysis. In this case there appears to be a combination of paralysis of the palate and pharynx, probably due to local absorption of toxin by the perineural lymphatics, and an acute ascending paralysis due to the general diffusion of toxin throughout the body. The position then, of the cases in Group II as cases of ascending polyneuritis seems to be secure. On the grounds that they differ from the usual case of multiple neuritis in their generalised and ascending character, their high mortality, and their greater incidence on men than on women, there is some justification for placing them in a group by themselves.

Group III is in some ways a little more difficult to place, but many of its features suggest a connection with anterior poliomyelitis. The average age incidence is definitely lower than in either of the other groups. The paralysis tends to appear after a variable period during which the only signs are those of an acute febrile illness, sometimes with a suggestion of meningeal irritation. The paralysis is not quite so regularly ascending and shows a tendency to appear in one limb first; further, in the two cases which recovered it was associated with wasting of the legs in one and weakness of one leg six months later in the other. Sphincter disturbance was not uncommon, but neither is it in the acute stage of anterior poliomyelitis. Sensory changes were slight, as would be expected, but there seems to have been an absence of the pains and tenderness in the limbs which occur often in anterior poliomyelitis. The cerebrospinal fluid in one case showed a pleocytosis. The post-mortem findings in one case were not essentially different from those described for acute anterior poliomyelitis (Buzzard and Greenfield, 1921); those in the other case published with a post-mortem report will be discussed later. The main differences from acute anterior poliomyelitis are as follows. The age incidence, although lower than that in Groups I and II, is yet high. The mortality is very high, and the extent of paralysis greater than in typical anterior poliomyelitis. Nevertheless, these are only differences in degree. There is some doubt whether all cases of anterior poliomyelitis are really the same in their

etiology and fundamental pathology, and it is not suggested that this group is necessarily homogeneous. They do, however, suggest from their clinical course an acute febrile illness, due probably to an infection and further, from the pathological findings, to an infection via the blood stream. Buzzard (1907) brought forward reasons for supposing that the infective agent in anterior poliomyelitis was blood-borne, and, on account of the similarity of the post-mortem findings, the same would seem to hold good for these cases. The other case with a post-mortem report, besides showing changes very like those of anterior poliomyelitis, showed also the presence of a bacillus, probably *B. anthracis*. Is it not possible that in this case, owing to some peculiar susceptibility obtaining at the time, the syndrome and post-mortem findings were caused by a septicæmia due to *B. anthracis* ?

Except that the signs and symptoms are rather less variable, this group is of the same nature as Group II, the variable toxin being replaced by a variable infection, with corresponding changes in the post-mortem findings. On these grounds the name 'acute infective ascending paralysis' is suggested for this group.

The position of Group I must now be discussed. There is no doubt that in these cases there is a poisoning of the lower motor neurone, leading to rapid loss of function. Whether this poisoning affects only the cell body in the spinal cord, or whether it acts as well on the axon, or even on the axon alone, is in the present state of our knowledge no more than an idle speculation. Buzzard (1907) states that in cases of acute toxic polyneuritis changes may be found in the anterior horn cells before they are evident in the peripheral nerves ; it cannot be concluded from this that these changes are necessarily secondary to an affection of the nerves which has not yet shown itself, although this is a possibility. Actually, the only point which distinguishes the cases in Group I from those in Groups II and III is the absence of reaction, whether constitutional or local. This cannot be explained by saying that the patients die before there is time for changes to become apparent ; the average duration of the cases was approximately the same as in Group II and longer than in Group III. The presence or absence of reaction depends more on the peculiar condition of the patient than upon the actual cause of the disease, so that Landry's ascending paralysis appears to belong to the group of diseases which depend chiefly on 'diathesis' or 'idiosyncrasy.' This is the conclusion reached by Grünwald in 1923. He further suggests that the condition may be an anaphylactic manifestation, brought about by the action of a toxin on a nervous system already sensitised by the previous action of the same or another toxin or infection. In this way he explains the common association of Landry's paralysis with a large number of different infective and toxic conditions. Exactly the same arguments would apply to cases of multiple neuritis, and at present the suggestion can be no more than a speculation. He also suggests the possibility of a constitutional diathesis, associated occasionally with the presence of such stigmata as hypertrophy of the thymus, an increase in the

number of corpora amylacea, islands of myelin in the grey matter, asymmetry of the horns of the grey matter, an open central canal, etc. Apart from the fact that the central canal was certainly open in the two cases reported in this paper with post-mortem findings, no support for this suggestion can be found in this series. It, too, must remain in the realm of speculation.

Whatever predisposing cause there may be for the development of the syndrome of Landry's paralysis, there must be some immediate cause. No light has so far been thrown on its nature. Prima facie, a toxin would seem most probable, but one cannot exclude the possibility of an infection.

There is another group of cases which is certainly allied to the cases which have been described, viz. acute myelitis. Clinically they are distinct, in that they show invariably marked sensory loss, usually affecting deep as well a superficial sensibility, and usually signs of pyramidal involvement. They have a similar etiology, being associated with chill, exposure, and acute specific fevers and syphilis particularly. There is also a toxic variety sometimes associated with pregnancy; these have sometimes been reported as cases of Landry's paralysis, e.g., by Marinesco (1924), who describes a non-fatal case of acute ascending paralysis, occurring during the puerperium, and associated with marked sensory loss, exaggerations of knee- and ankle-jerks, and a double extensor response. There is no need to describe the post-mortem findings of such cases in detail; they can be found in Buzzard and Greenfield's book (1921) and in Buzzard's paper, 1907. Buzzard himself considers that these cases are due to an acute infection of the nervous system of lymphatic origin, contrasting them with cases of anterior poliomyelitis where the infection is carried by the blood stream. It is possible that in those cases where no infection can be traced, e.g., the cases associated with pregnancy, the toxin is brought to the nervous system by the lymphatics. One is tempted to contrast these cases with cases of Landry's paralysis, suggesting that in the latter the toxin is conveyed chiefly by the blood-stream, or at least exerts its influence by that route, and that the former are similar except that the toxin exerts its influence through the lymphatic channels. It is perhaps significant in this connection that in one case an area of tuberculous meningitis was associated with the development of the typical syndrome of Landry's paralysis. An area of meningitis of this kind is more commonly associated with the development of a tuberculous myelitis.

SUMMARY AND CONCLUSIONS.

Cases of acute ascending paralysis can be divided into three groups; a poliomyelitic group, a polyneuritic group, and a group which shows little or no post-mortem change and no constitutional reaction. This grouping has already been suggested by Saltman, quoted by Pfeiffer, 1912.

These groups can be distinguished with more or less certainty on clinical and pathological grounds. A high mortality and a preponderating incidence on young adult males are characteristic of the whole series.

Although clinically and pathologically these cases fall into sufficiently distinct groups to be described as 'diseases' in the traditional manner, they have no common etiology. Landry's paralysis is a syndrome probably caused by a toxin exerting its influence through the blood-stream, and more or less closely related on the one hand to a group of cases which are simply a variety of multiple neuritis, and on the other to a group which are best described as cases of infective poliomyelitis.

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PSYCHOSIS ASSOCIATED WITH MYXŒDEMA.*

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HISTORY AND LITERATURE.

Gull, in 1873, presented a paper before the Clinical Society of London entitled "On a cretinoid state supervening in adult life in women," in which the chief symptoms of myxœdema were given. Ord, in 1877, presented material from observations collected since 1861 to the Royal Medico-Chirurgical Society of London entitled, "On myxœdema, a term proposed to be applied to an essential condition of 'cretinoid' affection occasionally observed in middle-aged women." A committee was appointed in 1883 by the Clinical Society of London to study the subject of myxœdema. Their report[14], a most interesting document, included a complete bibliography; it was published in 1888 and gave an analysis of all known clinical cases up to that time. Very little has been added since then to the purely clinical features and the elaborate tables of clinical data in 109 cases they were able to find. They summarized the intellectual changes, with notes pertaining thereto, as follows:

Slowness (report of 60 cases).—Absent in three. More or less marked in 56, doubtful in one. Of the intellectual changes, slowness in apprehension, thought, and action is most constant, its absence being noted in three cases only.

Abnormal persistence in thought and action (report of 29 cases).—Absent in 22. Present in seven. Abnormal persistence in thought and action is recorded in about one case in four.

Imperfection or perfection of mental processes (report of 41 cases).—Imperfection in 15, perfection in 26. In a rather larger proportion there is more or less imperfection of mental processes, the defect being, as noted before, one of retardation or sluggishness.

Writing (report of 33 cases).—Good in twelve, slow in seven, more or less inability in eight. Six were illiterate. (Specimens of handwriting were sent in four cases, but no opinion given). Writing is sometimes slow, sometimes imperfect, conditions which may in large measure be attributed to the state of the hands. In the case of educated persons the handwriting is usually good, and the length of letters, in all respects well indited, is remarkable.

Irritability (report of 53 cases).—Absent in 19. Marked in 32, placid but fretful at times in two. Irritability is a marked feature, though in exceptional instances the reverse is stated. In some cases placidity alternates with occasional outbursts of fretfulness or irritability.

Sleep (report of 73 cases).—Good in 37, bad and disturbed by dreams and sensations in 25, drowsiness during the day (both in good and bad sleepers) in 17, variable in one. In a large proportion sleep is noted as good, but in many of these there is excessive somnolence, especially in the daytime. In about a third of the cases wakefulness is recorded,

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and sleep is often disturbed by horrible dreams and sensations. It may be noted that drowsiness during the day is common in myxœdema in both good and bad sleepers.

Delusions (*report of* 46 *cases*).—Absent in 28. Present in 18.

Hallucinations (*report of* 43 *cases*).—Absent in 27. Present in 16. Delusions and hallucinations occur in nearly half the cases, mainly where the disease is advanced.

Insanity (*report of* 45 *cases*).—Absent in 29. Present in 16. Insanity, as a complication, is noted in about the same proportion as delusions and hallucinations. It takes the form of acute or chronic mania, dementia or melancholia, with a marked predominance of suspicion and self-accusation.

Agoraphobia (*report of* 33 *cases*).—Absent in 21. Present in twelve. Agoraphobia is present in a smaller proportion than the mental changes previously mentioned. In many cases its mere existence is noted, but, in a few, details are given, which show that this condition was undoubtedly present in a marked degree.

State of memory (*report of* 71 *cases*).—Good in 25, defective or bad in 46. Memory is usually impaired from an early period of the disease, especially in respect of recent events. It is recorded as deficient in 46 out of 71 cases.

Changes in speech (*report of* 104 *cases*).—Absent in four, marked in 100. It is evident from the tabulated returns that changes in speech are very constant. In four cases only, out of 104 replies, is no alteration noted, and in two of these four there is a history of slow and drawling speech in the initial stages. Slowness is the most marked feature, though in many cases articulation is described as either 'deliberate,' 'monotonous,' 'slurring,' 'hoarse,' 'nasal,' 'thick,' or 'hesitating.'

Eyes and vision (*report of* 83 *cases*).—Normal in 46, various impairments in 37. Various returns have been made under the head of 'eyes and vision.' In more than a half no abnormality exists. Most of the defects recorded are clearly accidental. In a small proportion two conditions are noted, which may probably be looked upon as belonging to the disease: (1) general diminution of acuteness of vision; (2) watering of the eyes. It may be mentioned here that exophthalmos has been observed once or twice in the early periods of myxœdema.

Ears and hearing (*report of* 60 *cases*).—Normal in 27, impaired in 32, one doubtful. Hearing is impaired in more than half, and in a considerable number the defect has been unilateral. As in the case of vision, it is probable that in some the impairment of hearing was simply coincident. Nevertheless, deafness to a varying extent must be looked upon as a condition proper to myxœdema, and not merely an accident.

Nose and smell (*report of* 54 *cases*).—Normal in 36. Smell impaired in eleven, perversion of smell in one, watery discharge from nose in six. The sense of smell is not infrequently defective. In a few cases a profuse watery nasal discharge is observed. In one instance perversion is noted.

Taste (*report of* 47 *cases*).—Normal in 27, defective in 18 (total loss in one, retarded in one, perverted in one). Taste is more often impaired than smell. In one case total loss is recorded. Retardation occurs in isolated cases only; but perverted subjective sensations are common.

Osler, in 1889, reported the unusual case of a man, aged thirty-one years, who had signs of exophthalmic goitre and of myxœdema with rapid pulse. Early in his illness he had ideas of being pursued. Later melæna and fatal delirium with glycosuria, in which he was maniacal, developed.

Hooper, in 1903, reported a case in which anorexia nervosa was a precursor of myxœdema. Régis, in 1911, reported the case of a woman, aged thirty-three years, with onset of psychic symptoms and myxœdema. She recovered when thyroid extract was given. The condition recurred on cessation of

treatment, and at the age of forty-five years she was sent to hospital with myxœdema. She had a mongoloid appearance and psychosis with chronic hallucinosis and confusion. Treatment helped the myxœdema but not the psychosis.

Bruce, in 1912, reported the case of a woman, aged forty-six years, in whom myxœdema and psychosis with loss of memory developed after exophthalmic goitre. She was disorientated, and thought friends would harm her and that her food was poisoned. She died with a high fever. Hæmorrhages were found in brain and spinal cord.

Barham, in 1912, recorded the case of a woman, aged thirty-four years, in whom "emotional conflict, alcohol, myxœdema and insane heredity" played a part. She recovered from psychosis twice.

Stanfield, in 1912, reported the case of a woman in whom myxœdema developed after much child-bearing, and who had excessive sexual desire. The patient recovered when extract of thyroid gland of sheep was given.

French, in 1918, mentioned, in a lecture to students at Guy's Hospital, two cases of myxœdema in women. In one case there was much somnolence punctuated with acute maniacal attacks; in the other there were headaches, vomiting, and somnolence, suggesting tumour of the brain for which trephining was done with fatal outcome. Necropsy revealed an extremely small amount of thyroid tissue.

Uyematsu, in 1920, reported the case of a woman with myxœdema and psychosis in which the patient was less active but was irritable, untidy, forgetful, somnolent and vertiginous. She wandered on the streets, was accusing, and threatened suicide. He reported a pathological study.

Claude and Bernard, in 1924, published the case of a woman, aged fifty-three years, with myxœdema, ideas of persecution, visual hallucinations and anxiety, who finally became depressed and self-accusatory. They felt that the myxœdema had a part in the onset of the disease.

Jacobi, in 1929, reported the case of a woman, aged thirty-one years, in whom, after thyroidectomy, psychosis developed; hebetude, staring, and catatonic-like features were associated. She heard music, had illusions, and presented a clinical picture resembling schizophrenia.

Kraepelin referred to myxœdematous insanity. Clouston, in 1904, described eight cases. Wagner von Jauregg, in 1912, expressed the belief that 15 per cent. of patients with myxœdema have psychosis. Curschmann, in 1918, and Deusch, in 1919, considered the relation of the climacteric to the development of myxœdema and reported, respectively, five cases and one case.

One may conclude from the foregoing that the various types of psychosis associated with myxœdema are not specific, and that they present a wide range of symptoms. However much one desires to ignore or minimize the significance of heredity, it seems fairly established that the qualities of siblings resemble each other more than do those from an unrelated group selected at random. Siblings also bear significant similarity to the attributes of their progenitors.

There are probably latent hereditary tendencies in all of us which undue circumstances may bring to the fore. The total ' make-up ' (personality*) of life-long standing may, by the confluence of conditions, be exaggerated rapidly, intensified, or replaced by another (latent) pattern of behaviour, sometimes to the bewilderment of the patient, his friends and associates. Although no effort is made in this paper to demonstrate the relationship of any certain type of psychosis to myxœdema, the latency and relativity of certain factors of hereditary and personal ' make-up ' are exemplified in the following cases.

REPORTS OF CASES.

CASE 1. A married woman, aged forty-nine years, first came to The Mayo Clinic in June, 1929, complaining of tiredness of six or seven years' duration. She always had been serious-minded, capable, quiet, good-natured, sometimes fearful, willing to take blame in situations which had not turned out properly and to which she was a party. A maternal uncle had been sent to an asylum following financial reverses. A brother of the patient at times had been very suspicious and had for considerable periods felt that attempts had been made to poison him. He had never been in a hospital. A brother had died of goitre. The patient had married a teacher when she was forty-three years of age and married life had seemed happy except for conditions to be recorded later. She had gone through the menopause eleven months before the examination. At the age of forty-three years, she had noted bulging of the eyes, tachycardia, restlessness, palpitation, loss of weight and strength, and other symptoms characteristic of exophthalmic goitre. At the age of forty-five years, she had been given sixteen treatments by roentgen rays to the thyroid gland. Following this, she had become " cold-blooded " (required room temperature of 80° F. to be comfortable); she was tired as before, and sleepy. Her legs ached; she gained weight, had a dry skin, œdema of the face and body, and was very sluggish.

The patient's condition was recognized as myxœdema and desiccated thyroid gland was given, but she felt that it caused abdominal distress and irritability, and she discontinued taking it for long periods. In the autumn of 1928, when the patient was aged forty-eight years, she began to feel that her husband was paying attention to another woman and that he was trying to do away with her by means of gas or the electric chair. During a game at a party in her own home during the holidays of 1928, she refused to sit in a chair designated for her, thinking it might be a plot to kill her. She also felt that her husband was trying to poison her and refused to take desiccated thyroid gland at home on account of such a belief. On several occasions subsequently, when desiccated thyroid gland was administered in sufficient quantity, at the repeated and urgent request of her physician, the delusions entirely disappeared and she felt so much better that she concluded that it was foolish to be taking medicine and discontinued taking it. On such occasions, the psychosis would slowly return in the same form as before. There were periods in which the psychosis was in evolution or dissolution when she was uncertain whether the so-called delusion was a product of her imagination or a real fact to be contended with. At the height of the psychosis she was always convinced of her husband's infidelity and attempts on her life by poison. At these times, she wished her physician to analyze her body or blood in order to detect the injurious poison or gas.

The facies and appearance were characteristic of myxœdema. The patient's weight

* Exemplified to some extent by such facts as may be elicited by the Amsden-Hoch personality outline.

was 193 pounds (the average normal weight had been 155 pounds); her skin and hair were dry; she had a bass voice; the pulse rate was 68; the systolic blood pressure was 120 and the diastolic pressure 90, measured in millimetres of mercury; the body temperature was 97·4° F. There was absence of free hydrochloric acid from the contents of the stomach after a test meal. The concentration of hæmoglobin was 77 per cent. (Dare); the Wassermann reaction of the blood was negative; examination of the ocular fundi gave negative results; old interstitial keratitis was present. Roentgenograms of the thorax, stomach, colon and gallbladder did not reveal signs of abnormality. Roentgenograms of the lumbosacral portion of the spinal column gave evidence of some hypertrophic arthritis. The few remaining teeth appeared to be infected. The thyroid gland was not enlarged. The basal metabolic rates were −40 and −34 per cent. Neurological examination gave objectively negative results except that the deep reflexes in the legs and at the tendons of the biceps brachii were like those seen in myxœdema (veratrine-like reaction). The patient was depressed. Thought and activity were much slowed. She felt certain of her husband's interest in other women and in his attempt to poison her. Her memory was adequate. She was completely oriented.

The patient was given 4 grains of desiccated thyroid gland a day. Within twenty days after she started to take the preparation of thyroid gland her delusions disappeared and she regarded them as foolish; the basal metabolic rate rose to −14 per cent. She continues to take thyroid gland and there have been no evidences of psychosis since she began to take it regularly. She calls her former delusions "foolish ideas and notions."

Summary of case.—A married woman, aged forty-nine years, about one year after the menopause, was in the depths of physiological sluggishness to which she descended in a state of marked myxœdema (after treatment by roentgen rays to the thyroid gland). She had a psychosis in which jealousy and fear of being poisoned were outstanding. She had a brother who is very suspicious and afraid of being poisoned. She recovered from the psychosis by taking desiccated thyroid gland regularly and has remained well for about a year.

Case 2. A married woman first came to The Mayo Clinic at the age of thirty-nine years, complaining of nervousness since the age of thirty-one years and marked depression since the age of thirty-six. Her father had died of diabetes mellitus and her mother had committed suicide in depression during the menopause. A cousin of the patient suffered from depression. The patient had always been sensitive but had had reasonable energy for work. She was interested in social activities, clubs, and so forth. There was no history of outstanding infection. At the age of thirty-one years she had begun to have tachycardia, exophthalmos, diarrhœa, tremor, and slight depression. She lost 20 pounds in weight. A diagnosis of toxic goitre was made several years later and nine treatments by roentgen rays were given to the thyroid gland. Her condition improved for six months, when the symptoms gradually recurred, except that the short attacks of depression, especially during menses, were more severe. Roentgen rays to the thyroid gland at the age of thirty-six years seemed to relieve many symptoms but did not relieve the depression as it seemed to have done previously. At the age of thirty-eight she gradually grew dissatisfied, and depression came over her, which was constant. She slept poorly. Removal of tonsils only aggravated the condition.

The patient had the sluggish, sallow, pasty skin and facies and slow speech of myxœdema. The pulse rate was 72 and the concentration of hæmoglobin 80 per cent. (Dare). The basal metabolic rates were −21 and −29 per cent. Neurological examination gave essentially negative results objectively. Her thoughts and activity were markedly slowed. She wept at times, and had the feeling that something dreadful would happen.

She had a feeling of being worthless. Hallucinations and delusions were not elicited and she was oriented.

When the patient was given desiccated thyroid gland, grains 2 to 4 daily, her condition improved, especially as to the depression, and in ten days the basal metabolic rate rose to —11 per cent.

Summary of case.—**A woman, aged thirty-nine years, whose mother had had severe depression, had short depressive attacks in the course of exophthalmic goitre; but when myxœdema subsequently developed severe, retarded depression appeared, which was considerably relieved by administration of desiccated thyroid gland.**

Case 3. A married woman first came to The Mayo Clinic at the age of fifty-eight years because of "nervousness, shakiness, and weak spells of a year's duration." Except that her father used much alcohol, relevant facts were not elicited in the family history. She had always been well, jovial, happy, talkative, and energetic, and despite her age, regarded herself as a young person. She was known as "the life of social gatherings in her community." She declared that she always could hear the slightest sounds and responded to them. She had always slept lightly but had never regarded herself as nervous until the onset of the present illness. She had lost 30 pounds in weight, perspired freely, and her lower extremities had become weak. She had worn less clothing since the onset of the present illness. She was dyspnœic on exertion.

The heart was rapid and palpitated. Impressions gained from the patient's history, appearance and basal metabolic rates of +16 to +29 per cent. led to a diagnosis of exophthalmic goitre, and subtotal thyroidectomy was done. The pathologist reported a hypertrophic, parenchymatous thyroid gland. She made an uneventful recovery and returned to her home, much improved, in slightly more than a month from the time she first came to the Clinic. She got along well for two months, when she began to feel cold. She had gained 40 pounds in weight, was sluggish, her tissues seemed swollen, her voice was husky, and she wished to sleep all the time. These were decidedly unnatural characteristics for her. A short time before returning to the Clinic she awakened early in the morning and thought she saw her daughter dressed in black standing beside an organ playing a dirge. She said it was like a dream. She stated that she had not been depressed. She returned to the Clinic five months after her first registration with obvious signs of myxœdema.

The patient weighed 196 pounds (average normal weight had been 157 pounds). The pulse rate was 80. Her face was swollen and puffy, her voice was husky, and she spoke slowly and was much quieter than was usual for her. Her hair was dry. Definite œdema of the arms, legs and face was present. The concentration of hemoglobin was 74 per cent. (Dare). There was moderate sclerosis of the retinal arteries. The basal metabolic rate was —29 per cent. Except for the type of deep reflexes seen in myxœdema, neurological examination gave objectively negative results.

The patient was given desiccated thyroid gland, grains 2, daily, and within a few days began to talk rapidly on any topic, jumping from one subject to another in rapid succession. On one occasion she thought she heard a radio, but no radio was near. She became restless and walked about in her room. Sometimes she would sing. She was very alert to every sound and visual impression, and any such stimulus might start a rapid stream of thought in which 'clang' association and the slightest associations in meaning set off streams of sentences (flight of ideas and rambling). She was not elated or depressed; she was oriented. When questioned, there was much verbal sparring and almost playful evasiveness. At times there was evidence of straining in the push of talk and activity, which were out of the ordinary. She declared that to maintain normal contact with the environment she continually had to make an effort in speech and activity. She felt that

her threshold of excitability had been lowered. As the basal metabolic rate approached normal she became quiet, and for about an hour one day she wept and said she felt lonesome. Subsequently she returned to her normal personality as described, and is in that state to-day, but continues to take desiccated thyroid gland.

Summary of case.—In a sociable, active, talkative, jovial woman, aged fifty-eight years, with hyperacusis and hypomanic disposition, myxœdema developed after thyroidectomy. During recovery, while she was taking desiccated thyroid gland, she became mildly manic, from which state she recovered to that of her normal personality when the basal metabolic rate became normal.

COMMENT.

In the first case it might be said that a latent hereditary disposition to suspiciousness and a paranoid trend were brought out by myxœdema and disappeared when the latter passed away. In the second case a tendency to depression, hereditary or constitutional, came out strongly with myxœdema. In the third case a mildly manic constitution was accentuated to a manic reaction without elation, while the physiological depression of myxœdema was being removed. All patients improved on the administration of desiccated thyroid gland.

Not all myxœdematous patients have depression, elation, or overactivity, nor is myxœdema an associated state in many patients who have a similar psychosis. However, thyroid deficiency appears to have had an effect of bringing into relief latent hereditary disposition and constitutional tendencies. One may conclude from the forms of psychosis associated with myxœdema that different patients react differently to the same physiological depression. Further excursions into the nature of the intimate processes of life (thought, action, and emotion) need to be made when the metabolic fires burn low. These unusual cases are so interesting that conditions, drugs, circumstances and insidious biological changes which might in any way simulate the effect of myxœdema are worthy of much study in order to understand the strata of latency which should be guarded against, as well as some which might (if unearthed) be of inestimable value to some otherwise chronically incapacitated persons. What can be done that may be comparable with the administration of desiccated thyroid gland in other types of psychosis not unlike those associated with myxœdema but not related to it? That there seems to be a correlation between physiological depression and the state of one's affects is apparent in not a few cases. That there is physiological depression, certainly at times, during many cases of psychosis also is apparent. The preceding observations again emphasize the necessity of avoiding the over-specialized study of patients, especially classifying them into 'mind and body types.' Persons who are distressed and incapacitated should be studied from every biological standpoint to ascertain the assets and liabilities which make for the presence or absence of health, vigour and usefulness. By such comprehensive regard for patients, the factors which help them to recover those feelings and capabilities with which they can be most comfortable and usefnl may be discovered.

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CONGENITAL WORD-DEAFNESS, WITH SOME OBSERVATIONS ON THE ACCOMPANYING IDIOGLOSSIA.*

By

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I EXAMINED Wilfred C., age 13 years, at a routine school medical inspection. His speech had always been most indistinct; and he had been thought to be very deaf. Such had been the excuse for his scholastic backwardness, for his attainments in school subjects are most limited—practically entirely to arithmetic and to writing and drawing from copy. He quickly informs one, 'I cannot read.' Yet he is recognised as one of the most observant boys in the class; his conduct is perfect; and he acts as the class monitor—indeed, is one of the best the teacher has had.

He had been thought to be very deaf. It has always been noted that he will pay no heed to conversation unless his attention is attracted very forcibly. At the examination a watch was held at the usual distance from his ear, and he was asked 'do you hear that?' The lad seemed only to mutter something. He was then tested by the spoken voice, when facing at right angles to the observer. He did not answer such questions as 'what is your name?' 'what school do you attend?' nor did he obey such commands as 'point to the door.'

He cannot go errands because 'he cannot pronounce words correctly.' His teacher admits that she always has the greatest difficulty in following his speech, and very often finds it quite impossible to do so. Examination revealed indeed a strange kind of language.

This patient has been more thoroughly investigated. It is to be noted at once that the observations have been difficult to make. For example, it was found necessary to use his ability to copy in order to test visual acuity by Snellen's types; and much repetition of simple sentences has been necessary for the boy to comprehend the meaning of the examiner, and often much repetition has been demanded from the boy for the examiner in turn to comprehend him.

(1) He hears many, if not all, sounds with normal acuteness. When blindfolded he named correctly the number of soft peals made with a hand bell, and the number of snaps made with the fingers. He repeated after the examiner, one by one, the letters of the alphabet and such familiar words as 'mother,' 'father,' 'me,' 'pen,' and it was the recognition of the fact that he reproduced some of the sounds in some of the words spoken to him

* I am indebted to Dr. Paget Moffatt, Medical Officer of Health, Bolton, for permission to publish this paper.

when his hearing was being tested, that suggested a 'word-deafness' to the examiner. He recognised and named the mew of the cat, the bark of the dog, and the low of the cow. He was also able (when made to understand what was required of him) to add and subtract in units, and to solve such simple addition as 3s.+4d.+2d. But with the eyes covered he was evidently quite unaware that he was being asked a question, or being requested to perform any action, e.g., 'what is your name?' 'who is your teacher?' point to the door.' He only repeated a few sounds bearing, on occasion, a resemblance to the sounds spoken to him. He did not understand even his mother and his teacher.

When however the eyes were freed, and he was allowed to observe our faces, he comprehended to a degree. He understood his younger brother, who is in the same class at school, most easily and best, and his teacher better than the examiner. He answered correctly simple questions as 'where do you live?' 'can you read?' and recited in his own sounds the days of the week and the months of the year; and on request pointed out, in a picture, a basket and a windmill, mewed like the cat, barked like the dog, and lowed like the cow Indeed, it had been only by freeing the eyes, explaining what was required, and blind-folding again that the observations recorded above concerning acuity of hearing were made.

(2) He quickly stated, 'I cannot read.' He did read correctly 'A,' 'B,' and 'the'; 'C' was called 'T'; 'HOME' was 'TONG'; 'CAT' was 'AS'; and nothing was made of 'DOG' or of 'MY MOTHER.' But '184,' '263,' '1,843' were read correctly and quickly—e.g., the last as 'one thousand eight hundred and forty-three,' and '£384 10s. 9d.' was read as 'three hundred and eighty-four pounds ten shillings and nine pence'—*after he was told it was 'money.*' He read correctly the time from a watch.

(3) When asked to repeat[1]: (i) 'In summer the sun is hot'; (ii) 'I have a little dog'; (iii) 'The dog runs after the cat,' he said, with the eyes open, (i) 'In tummer der doo doh'; (ii) 'I have e u doze'; (iii) 'The don run haffy'; and when blindfolded (i) 'In tummer'; (ii) 'I have e eum don'; (iii) 'To don one tat.' (The question arises whether he was saying in his own way 'two dogs, one cat'). He repeated correctly in order five numbers[2], both with the eyes open and closed.

(4) He named at sight such objects as a knife, pen, pencil, and in a picture a basket and a teapot. He named too, from memory, some of the boys in his class.

(5) He is very quiet—practically never talks unless he has been addressed, and when he does talk all have the greatest difficulty in following him. Often his utterances are quite unintelligible.

(6) He transcribed correctly with correct punctuation marks in a very good cursive hand the following printed passage[3]: 'I have something in my pocket which I use to tell the time, do not say what it is called; but tell me how many hands you think it has.' But neither could he read it aloud, nor did he understand the passage. He could not write to dictation. He was asked to write, 'get me a pen.' He was allowed to lip-read; he repeated the sentence aloud; he performed the act and then wrote only something like 'men B.' He never writes composition at school, but is allowed to set himself sums or to draw from copy. When requested to write something he asked, 'sums?' and then 'money sums?' and wrote the following:—

£	s.	d.
16	7	8
16	1	9
19	6	9
51	16	2

(7) He is said to be fond of music. He recognised the examiner's whistling of 'God save the King.' He sang on request the hymn with which the school is opened daily.

(8) He named correctly a knife, penny, shilling, key, and indicated by gesture that he recognised a rubber name-stamp, when these were placed in his hand.

In the above description, only in (3) and (5) has any notice been taken of the speech. But the boy has a language of his very own. I have heard the following: 'Saturday night' is 'Tatur nigh'; 'two comics' is 'two commas'; 'July' is 'Julyk'; 'August' is 'Angus'; 'September' is 'Temtember'; 'quarter-past' is 'tarter tast'; 'basket' is 'bassie.' Of the letters, 'C,' 'H,' 'J,' 'Q,' 'W,' 'X,' 'Z,' are pronounced as 'T,' 'S,' 'Tae,' 'Jew,' 'Dub,' 'S,' 'Shade.' His recitation of 'God save the King' was a true idioglossia, commencing something in this wise 'God tave on able it, Bot tave ta ti.' His singing of the morning hymn and his description of a scene at the pictures (a fight with a boy and a girl) seemed to be a conglomeration of gibberish. But certainly he remembered the movements of the picture perfectly well, for his accompanying gestures proved an orderly sequence of thought, although his language was indescribable.

On routine clinical examination, the heart and palpable blood-vessels are found to be normal, as are both ear-drums. The nervous system is normal. He is right-handed; the eyes externally, fundi, optic discs, visual fields and acuity of vision, superficial and deep reflexes, co-ordination, and sensation to touch cotton wool and pin prick are all natural. He is neither feebleminded nor antisocial. The expression is intelligent, his conduct is excellent; he is happy and contented and a favourite in the class. He understands the value of money. He quickly solved simple zig-zag puzzles, and was as smart as the examiner at the game of 'snap.'

DISCUSSION.

Thus there is described the case of a boy who hears although he does not comprehend the meaning of words by hearing—a boy, it seems, who comprehends spoken language only to a degree, and only by lip-reading or by accompanying gesture. Though he understands through the auditory channels music (admittedly only roughly tested), familiar animal calls, and numbers, yet the simplest sentences of the simplest words are not comprehended through the unaided auditory sense; nevertheless the acuity of hearing seems itself not at all defective.

Sight and visual memory seem to be normal. For we have found that acuity of vision is normal, that he names objects at sight and from memory, and further that it is only when he is allowed the use of his eyes that he understands the speech of another. His inability to read anything but numbers need not be due to any inherent defect in a visual memory sense, for he can copy from print to cursive handwriting, he can name objects at sight, and (this is most pertinent) he enjoys and remembers the cinema (a marked contrast to his indifference to a 'speech' or a 'story'). He buys children's comics too, and follows their little stories from their numerous illustrations. This inability to read is probably due to the fact that any attempt to teach him reading has been through the auditory sense—a sense through which we have seen comprehension of words entirely fails. (From an experiment just begun upon normal school children it almost seems possible to make a comparative measurement of a visual memory alone (for letters), and Wilfred's attainment corresponds to a normal average as found so far.)

I suggest that the condition revealed in this boy is explained by postulating a marked defect, or, best of all, an absence, of an auditory word-memory,

while there is present an auditory memory for numbers and for some other sounds, at any rate. The defect seems pure, for examination reveals a normal 'make-up' in all other ways except that there has been educated by self-training a new memory (say 'centre') as named recently by Worster-Drought and Allen[4] 'the lip-reading word-memory centre.' That this 'memory' is still incomplete and slow of function is obvious because of his failure to comprehend much of, and his difficulty to comprehend at all from, the speech of others, and from a few mistakes the boy made. He was set this adding sum :—

£	s.	d.
6	8	10
248	16	4

and he wrote as his answer 254 25 4. He insisted he was correct until with difficulty he was brought to understand it was a 'money sum,' when he quickly altered his denominators and gave the result as £255 5s. 2d. Again he was heard to say 'seven and four leaves eleven' when it was quite appropriate for him to make eleven by the addition of seven and four. His scholastic achievement is arithmetic; he can add, subtract, multiply and divide in numbers; he can add in money but not in avoirdupois, and he cannot do the simplest problem because, apparently, of the incompleteness of this lip-reading word-memory. His strange speech, too, seems to provide evidence of the incompleteness or slovenliness of its education. These points seem to be proved later by consideration of the state in which we found the 'lip-reading word-memory' of his brother Tom.

ETIOLOGY.

Let us now consider the etiology of this auditory word-memory defect of Wilfred C.

We have seen the parents and all five sons of this family. The parents are of normal intelligence for the labouring class to which they belong. Of their own family histories they will admit nothing abnormal. They both attended ordinary schools and can converse normally. The eldest son progressed normally at school, as the fourth is now doing. The second son, Tom, attended a deaf school for seven years. Wilfred is the third son. The fifth boy, James, is now six years old, and had an indistinct speech on entering school.

During the pregnancy his mother was perfectly well and had no accident. The birth was at full term, a midwife was in attendance, there being 'no need for a doctor,' and Wilfred was a big fine baby. He seemed to his mother to develop normally—sitting up at six months, walking at 18 months (he was very fat) and saying 'Dad,' 'Ma-ma,' and 'butty' ('bread and butter') at two years. But he was a quiet infant and did not cry much, although the cry was not noted as exceptional in character. As he grew older, he "would not bother

with other children," but would be always with his mother, who came to think of him as just "so very quiet," but always as "sharp enough," until she was told the teacher "could not make him understand at school." He has always liked running about in and about the home, has been fond of boyish mischief, and now likes to make things. He makes boxes for his mother, and 'bicycles' from old wheels for the boys of his street. He enjoys very much going to the pictures. Apart from diphtheria at 14 months and two operations for the removal of adenoids he has had uninterrupted good health.

His brother Tom was sent to a deaf school when eight years of age. Tom is more easily examined than is Wilfred, for he understands the examiner more quickly and his language is more easy to follow. But his general intelligence is not so good as that of Wilfred. He did not score well in his efforts at zig-zag puzzles, nor did he comprehend so smartly a card game of 'snap.'

He heard the watch at four inches right and left ears, both with eyes open and closed.

He can read; he tried all the words up to age 12^5. He read aloud in a monotone, and understood the passage 'I have something in my pocket' already given to Wilfred, for he answered 'two hands' and later said 'clock.' He repeated the test sentences as (i) 'In tummer the ton is tot'; (ii) 'I have the dog'; (iii) 'The dog ran after cat.' He named familiar objects at sight. He can copy. He wrote to dictation for (i) 'I live with mother and father,' (ii) 'I like the pictures,' (i) 'I live which mother and father' and (ii) 'I like to picture.' His speech is of more ordinary pattern than is that of Wilfred. It is best described as very marked lalling—e.g., his repetition of 'in summer the sun is hot'; his addition of 'two and six and a penny' was 'two tix an penny two teven,' and a 'key' was named a 'king.' But when Tom was blindfolded or the examiner went behind him, he did not understand what was said to him. He was asked to sit down on a chair and he answered by pointing to his father. He did not understand 'take off your cap,' or 'stand up,' or 'are you going to the pictures?' But such requests were promptly executed when he was allowed lip-reading. Tom, too, then, has very little, if any, auditory word-memory. I tried to teach him the meaning of the words 'stand up.' By much repetition of the sounds and action he seemed to have learned it. I then asked him, when his eyes were closed, to point to the door, then to put his cap on the floor, then to 'stand up,' and he failed completely. Again, when he was blindfolded 'I like the pictures' was said to him. On the eyes being freed he wrote 'I like to six.'

Tom was taught by the ordinary routine methods of a deaf school—by lip-reading. The result has been to develop in him a 'lip-reading word-memory' much in advance of that developed in Wilfred, who has had no such educational advantages. Because of this new memory he can talk in a way much more intelligible to others, and he has been taught to read; but still he has a defective auditory word-memory, although he has acuity of hearing practically normal. His auditory word-memory is not defective because of any lack of its education; it has been bombarded by every known means, and it has not responded. It must be inherently defective if not entirely absent, a condition of affairs similar to that revealed in Wilfred. His only illnesses have been two operations for the removal of tonsils and adenoids.

DISCUSSION.

In one family then, we have found two brothers suffering from a congenital absence or grave defect of an auditory word-memory. There is no other defect or lesion which can be demonstrated; indeed, in Wilfred the lesion is singularly pure for words. There is neither intra-uterine condition nor birth

injury nor early illness to account for the condition; and even were there some such unrecognised cause, it seems reasonable still to believe that there must be an inherent familial weakness in this 'memory,' for it alone, from any cause, to have thus suffered in two members of one family.

Any cerebral localisation of this word-memory defect, from a consideration of the cases of these two boys, has been impossible, if the defect be indeed a local one. Tredgold[6] writes: "Considered psychologically, both word-blindness and -deafness are probably due to particular defects in the capacity for symbolic imagery. The patients are neither blind nor deaf in the ordinary sense, but something is lacking in consequence of which the *meaning* of certain characters, the idea they stand for (alphabetical or numeral) or, in the case of the word-deaf the meaning of particular sounds, fails to be apprehended. Doubtless this has a material basis in the imperfect development, or inborn deficiency, of certain groups of neurones, but the actual site of this has not yet been determined." Our two cases take us no further than this as regards any definite 'localisation.' The condition may perhaps be due to some 'departure from the normal' either in the function of some specialised cerebral cells, or of the brain's general intricate and harmonious working as a whole.

But consideration of these two cases throws some light on the condition of idioglossia as found in Wilfred. He has always been quiet, would as soon not talk as talk, indeed he seldom does speak unless first spoken to. But he is an intelligent and observant boy. He has all his life been hearing his associates accompany their actions and exchange their thoughts by sounds. He has found it expedient in his life amongst others at home and at school to develop and to use a lip-reading word-memory and he, too, has come to clothe his thoughts, as others do, in words. But his lip-reading word-memory is, in development, still far behind his capacity for rational sequence of thought. It seems to be just so far behind as his idioglossia is different from the glossia of his normal associates. For as he educates this memory of his, his own sound-cloak for thoughts, he will be wise enough, according to his intelligence, to replace it by a cloak which will be as similar as he can make it to that used by those by whom he wishes to be understood. The idioglossia as we have found it, then, would appear to be the result of a partially developed lip-reading word-memory being accompanied by a normal acuity of hearing.

The speech of these two boys is not at all like that of the 'congenitally deaf' seen in deaf schools. In such children there is no idioglossia, but only a mechanical reproduction (in monotone) and use of the teacher's words according to the completeness of the speech education obtained—and there is no more. Tom's reading is of this same character and is in monotone. Wilfred, however, is continually hearing sounds made by others, sounds which he has found he can reproduce; but he has taken to sounding his thoughts in his very own way, for he has not word-memory enough with which to come to understand the meaning others give to any particular words. As we have

said, the purity of his idioglossia will remain until it is replaced by the fuller education of his lip-reading word-memory—for then he will realise he will possess 'counters'[7] for the exchange of thought of considerably more value to himself. On the other hand, Tom has had educated, to a marked degree, this lip-reading word-memory and through it he can read (like the deaf in a monotonous speech); but he has acuity of hearing which gave him at first an idioglossia, an individual language which he has gradually replaced as he has benefited, according to his general intelligence, by his special educational advantages. This would explain the observation of Tom's teacher at the deaf school—"We got him to talk much better, for at first his words were a jumble, but if only he had not talked by himself but left us to teach him, he would have learned to talk much more distinctly."

The headmistress reports it has been most difficult to teach James to read, but success has now come after much individual and oft-repeated tuition, and he now talks more distinctly. On examination, he follows language spoken from behind. He is not, then, word-deaf, but it seems that he has had a relative difficulty in learning the meanings of sounds, and that he has had a relative 'idioglossia' which he is now losing as his education is advancing.

Finally, it is striking how happy and contented these two boys appear to be. Wilfred, it is true, cried the first time he was blindfolded, but it was not clear whether this was not from apprehension rather than from any sense of inferiority. Later, he seemed only pleasantly amused at the examination, which gradually revealed his affliction. Neither of the boys seems to be aware of any gravity in their disability. Wilfred tells quite with satisfaction that he is going to work in a mill when he leaves school—'work for money.' Any thought of handicap evidently has not struck him at all. Perhaps this is because he has not lost something; rather, he has gradually been adding to his accomplishments. At any rate handicapped these boys will certainly be in their common association with others—handicapped, misunderstood and ill-understood probably more than if they had been deaf-and-dumb, for though hearing they will be found not to comprehend, since to hear aright they always must see.

SUMMARY.

(1) A case has been described, similar to those which have on rare occasions been reported as of 'congenital word-deafness.'

(2) In this instance the condition has been found to be familial—two brothers of one family being so afflicted, and a third relatively so.

(3) If this condition be due to a lesion of some anatomical entity—an auditory word-centre—the usual methods of examination as have been employed in these two cases do not shed light on the cerebral localisation of such a centre. All that can be said is, that, if there be such a centre, it must be of very great specialisation.

(4) It is suggested as possible that the condition may be due to an unusual form of the functional harmony (as an intelligent whole) of the cerebral 'make-up' of the individual.

(5) The results of, or social handicap due to, such a grave inherent abnormal condition appear to depend for the great part on the general intelligence of the afflicted, as measured by his capacity to respond to the educational advantages he may have had.

(6) The fact that one brother has been taught in a deaf school by methods similar to those used in the education of the congenitally deaf allows certain observations to be made which seem to shed light on the phenomenon of idioglossia.

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BIOLOGICAL AND SEROLOGICAL METHODS OF DIAGNOSIS IN EPILEPSY.*

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Pathologo-humoral methods of research which have been pursued in epileptic patients may be useful in three different respects : (1) The results and obtained relations inform us of the beginning of the disease and of the way the epileptic attacks occur. (2) They may be used for causal treatment. (3) They offer a convenient method, on a pathologo-humoral basis, for the diagnosis of epilepsy.

In all three respects these humoral and pathological researches have given valuable results. Researches on the phases of epilepsy have also led to essential conclusions. They show the modifications which appear in epilepsy during periods where no symptoms are to be observed, as well as in relation to the attacks.

The study of metabolism shows that in the processes of oxidation there appears a decrease compared with the normal. Experiments with carbohydrates and albumin show a relative decrease of humoral activity and of the production of carbonic acid. The azotic exchanges prove a modification related to the epileptic attack. Before the paroxysms one finds the smallest values of nitrogen, and after the attack it rises again. During the attacks one observes nitrogen retention, which Allers[1] attributes to circulating albumin. As for exogenous purins, the study of metabolism (as Rohde[22] has shown) proves a slower combustion of these. One may observe a diminution in the rate of excretion of urinary acids. Some observers mention also a toxic condition of the nucleoproteins.

The alterations of the elements which occur in epilepsy naturally react upon the state of the body humours. Chemical, biological and morphological blood analyses also show alteration, which Di Gaspero and De Crinis[9] have first demonstrated to occur. *If we sum up the humoropathological characteristics of the blood serum, we see that the albumin quotient of the serum in epileptics offers greater fluctuations than in normal persons.* Before the attacks the albumin content goes rapidly up, reaching its highest point immediately before them ; afterwards it falls suddenly. The rise and fall of the tension of blood shows an evident parallelism with the oscillations of the albumin. The time of coagulation may also fluctuate, and, in particular, be longer before the attack. Di Gaspero has also noticed leucopenia before the attack, and, on the contrary, leucocytosis during the attack. Further, one observes a rise in hæmoglobin

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and in viscosity as well as a decrease in the specific gravity of blood during the epileptic attack.

Bisgaard[4] observed in 30 genuine epileptics a decrease of the percentage of H–ionic concentration and also of NH_3 excretion, whereas he failed to find a similar decrease in 150 normal people. Disturbances of that kind are well known in tetany and are disclosed by the dosage of parathyroids. From this point of view Bisgaard considers that tetany and epilepsy are due to functional alterations of the endocrine glands. Bolten[5] also considers epilepsy as due to insufficiency of the thyroid and parathyroid glands. Pfeiffer[19] and De Crinis have also found a rise of the antitryptic ferments in blood serum. According to Bruehl[7] the proportion of creatines shows diminution, and, with the attack, higher values.

Among lipoid substances the quota of cholesterin in serum offers fluctuations during the attack similar to those of albumin. These facts deserve particular notice as the central nervous system is the chief storehouse of the lipoids, especially cholesterin ; and in epileptic attacks alterations of the central nervous system are to be put in the first rank.

Cuneo[8] has observed that the serum contains toxic albuminoses, especially before the attack. Many authors consider the pathological basis of epilepsy as the effect of these toxic substances contained in blood. Pfeiffer[19] has also shown the rise of parenteral albumin as a cause of epilepsy. For this reason, it may be assumed, the disease belongs to the class of toxic anaphylaxis.

Humoral alterations of the serum, observed during the attacks, occur indeed by themselves, without any attack. We term these the 'serologic equivalents' of the epileptic attack. It is very important from a practical standpoint to utilise humoral and pathological methods for the diagnosis of the disease.

This proof of the presence in the epileptic organism of toxic substances may be used for the diagnosis of the disease from a biological point of view. Of the chemical constitution of those bodies, acting toxically and appearing in body fluids after the epileptic attacks, we know nothing for certain. Some authors from experiments upon animals call in question the toxicity of the urine after the epileptic attack. Loewe[16] asserts strongly a relation of toxic elements of the serum to a substance in the urine which cannot dyalise and contains nitrogen. This substance, which he calls 'pesotexine,' is excreted after the attack in great quantity in the urine and can produce, according to his experiments, epileptic seizures in animals. These experiments do not however show a sufficiently coherent result. Examinations of the urine evacuated by catheterization and inoculation of epileptics' urine in animals are of no help for the diagnosis of epilepsy ; they have no significance since toxicity of urine is observed in many other psychoses. And furthermore those methods do not give results during the periods free from attacks, when it should be most desirable to make a diagnosis.

The explanation of urinary toxicity after the paroxysms is differently accounted for. Noervig[17] asserts that according to his experiments the epileptic organism presents disorders in the regulation of the H-ion concentration in urine. De Crinis points out that the central nervous system in the epileptic loses the power to regulate the 'acid-base equilibrium.'

There is far less discrepancy, if instead of inoculation by urine, animals are inoculated with epileptics' blood serum. During last year I had occasion in our clinic to pursue such experiments. We have worked out a biological method principally for the serological diagnosis of dementia præcox.

On the basis of the technique published in 1911 by Benedek and Deak[3], our aim was to study the degree of formation of immun-hæmolysin in rabbits which had been inoculated with blood serum of patients suffering from dementia præcox, general paralysis, epilepsy and some other nervous or mental diseases, and also the blood serum of healthy persons. So far we have three series of experiments and have inoculated 36 rabbits in 136 cases with blood serum of different patients, using either the venous or the intraperitoneal route. The number is explained by the fact that we have many times taken blood from the same person and used it for inoculation.

Our researches, principally the inoculation of blood serum of patients suffering from dementia præcox, led us to interesting results.

In our experiments we have evaluated the content of immun-hæmolysin of the blood serum of rabbits, as follows. One part of the immune serum was made inactive by leaving it 1½ hours in a water bath at 56° C. Each rabbit serum was used in that way to obtain two distinct series: active and inactive. We filled up 1 c.c. with an 0·85 per cent. NaCl solution, the dose of immune serum increasing from 0·1 or 0·2 to 0·5. Afterwards we added in the active series in each separate test tube another c.c. of the 0·85 per cent NaCl solution. In the active series we took only 1 c.c. of the 0·85 per cent. NaCl solution and ½ c.c. 10 per cent. fresh complement. In the two series we placed in each test tube of each group (in which were 5 tubes) ½ c.c. for every separate test tube of a 5 per cent. emulsion of washed red blood corpuscles—to the first group, of normal persons (or in some cases of epileptics); to the second, of patients suffering from dementia præcox; and to the third group, of patients suffering from general paralysis.

We obtained the 5 per cent. red blood corpuscles under the usual conditions, from fresh blood taken by venous puncture and defibrinated after careful washing with physiological NaCl solution. We kept the glass tubes of all groups in the incubator at 37° C. during two hours, and the result was usually read next morning when the series had stood for 10—12 hours at room temperature.

The normal content of hæmolysins in blood serum of rabbits as proved by our experiments is very small, and capable only in the highest titre of producing weak hæmolysis. The serum of the various rabbits shows only small deviation with reference to normal hæmolysin content. In spite of that

we have evaluated in each case the hæmolysin content before the injections, in order to compare this afterwards with the titre of the immun-hæmolysin attained after repeated injections. The technique in those inoculated animals was in some details variable. According to our researches the best method is to inoculate the rabbits intravenously with the serum of patients, performing the injection at intervals of four to five days. We always repeat it three times with doses of 3—4 c.c. The fifth day after the last injection, we take the blood of the animal by heart puncture and we again examine the immune serum with the different emulsions of red blood corpuscles in each of the described hæmolytic systems.

In all three groups of our experiments constant differences showed themselves between the results—according to the formation of immun-hæmolysin—of the inoculation on the one hand of serum from cases of dementia præcox, and on the other of serum from general paralytics or other mental patients, or, lastly, of healthy subjects.

For dementia præcox it may finally be accepted as a specific and special humoral characteristic that the inoculation of serum of patients suffering from that disease gives the highest titre of immun-hæmolysin formation in inoculated animals. We cannot describe that occurrence by a better name than ' positive pleohæmolytic reaction.'

During the ' pleohæmolytic ' researches we have used as a control method the blood serum extracted from 10 inoculated epileptics. We have injected in the ear-vein of rabbits 3—4 c.c. of blood serum. In six cases the animals were seized with tonic and clonic attacks from a quarter to one minute after the injection and three died during the seizure. The other inoculation of epileptic blood serum in animals was always intraperitoneal. In this way we did not obtain proof that the toxic blood serum could cause the attack, but only that after a series of injections the body-weight would notably decrease. Similar phenomena were observed in 15 cases published in 1921 by Pagniez, Mouzon and Turpin[18]. They inoculated rabbits in the heart with 2—4 c.c. of blood serum of epileptics. In three cases they produced a seizure. If the blood serum is warmed for ten minutes at the temperature of 58° C. it loses its toxic power.

The above-mentioned biological methods (with blood serum inoculations) may also be applied to the diagnosis of uncertain cases. From another point of view Trevisano[24] has employed the inoculations of serum for this purpose. He utilised anaphylactic shock in his experiments to prove epileptic causation. He first inoculated guineapigs with blood serum of normal subjects and some days after with spinal fluid from the same. No anaphylactic symptoms were observed. The same experiments pursued with blood serum and spinal fluid of epileptics bring anaphylactic symptoms into evidence. The animals exhibited general trembling, seizures, and decrease of the body temperature ; and in many cases death ensured. Identical effects appeared when first spinal

fluid and then blood serum were inoculated. The results of these inoculations do not seem to be influenced by the matter of whether spinal fluid and blood serum obtained immediately after the attack are used, or those of some time after.

Of the toxicity of epileptic spinal fluid we know nothing for certain. Kafka[14] tried to solve the question by well-considered experiments, but reached the same conclusion. Experiments were also made on the cytological, biological and chemical character of the spinal fluid and fluctuation in its pressure. The results of various investigations on the spinal fluid spectrum (Eskuchen[11]) or on the so-called 'humoral syndrome' in nervous and mental diseases have given very poor results for epilepsy. Our own researches, using epileptic spinal fluid collected during the attack as well as after it, did not demonstrate anything abnormal with the various methods of research already known. An increased spinal fluid pressure is usually found, especially during the attack. These observations had already been made by Redlich and Poetzl[21] in 1919 and afterwards by Boveri[6]. We have observed it ourselves in one-third of all cases. Suboccipital puncture showed in general that in the sitting position the spinal fluid pressure in the cisterna magna was (even during the periods free from attacks) positive, contrary to the normal negative pressure. In these cases the spinal fluid on puncture drops out by itself, when the patient is sitting.

Mention should be made of the evidence furnished by Donath[10] that the spinal fluid collected during the attack contains choline. Relying on this, he thinks that choline plays a part in the causation of the epileptic attack.

Notice also should be taken of the fact proved by Laures and Gascard[15] that whereas the urea content of spinal fluid increases during the epileptic attack, it decreases during the hysterical seizure; and this may be used for differential diagnosis. Wittgenstein[24] has observed hyperglycorrachia immediately after the attack. Recently Jacobi[13] has shown disorder of the 'interferometric rate' in the spinal fluid of epileptics, mostly an increase. Altenberger and Stern[2] assert that a secretion of the retrohypophysis is to be found in the spinal fluid. It was absent at the beginning of 79 cases of epilepsy, whereas they had found in 71 cases out of 80 of non-epileptics an average percentage of 0·00058 in the spinal fluid taken from the cisterna magna or of 0·00087 in that from the ventricles.

In clinical practice as regards the humoral-pathological diagnosis of epilepsy the above researches cannot be very useful as they depend upon difficult chemical analyses.

Hartmann and Di Gaspero[12] with methods used in clinical practice, found in epileptic cases a notable decrease of the albumin content of spinal fluid (·04—·06 per cent.) (Nissl-Esbach method). On the whole the chemical features of the spinal fluid in epileptics do not show anything certain when examined with methods already known. De Crinis stresses the fact that

"nothing characteristic is shown by the spinal fluid chemical standard in epileptics."

In our clinical laboratory, out of 5,780 spinal fluid examinations since 1922 to the present day, 332 concerned cases of epilepsy. We have observed always an absolutely negative 'spinal fluid spectrum.' Among colloid reactions of greatest sensitivity, the normomastic and the shellac reactions, and also the bicoloured mastic reaction[23], have in some instances given very evident deviations.

We have described the whole humoral syndrome on the basis of the so-called 'five reactions,' summarizing our researches—numerous enough—upon epileptic spinal fluid. These include the Wassermann reaction of serum and spinal fluid, cytological analysis, globulin and colloid reactions. We have also compared the results of these various methods. Our results are shown in Table I. A slight pleocytosis in epileptic spinal fluid occurs only in a small percentage of cases. The globulin and goldsol reactions seldom exhibit departure from the normal, and that is little. The highest percentage of these mild deviations is shown by the bicoloured mastic reaction, in the form of slightly irregular curves. *For that reason this colloid reaction may be regarded as the most precise among them all.*

The numbers between parentheses in the Table refer to cases where a small quantity of blood in the fluid or a reaction due to intervention (air or lipiodol injection) has modified the findings, or where the examined epileptic exhibits a seropositive latent syphilis. The great value of the bicoloured mastic reaction in the range of the '5 reactions' is shown also by the fact that in 16 cases we have found a feebly positive reaction proved by slight irregularity of curves in patients who showed also a seropositive latent syphilis.

After two years' research we have in our laboratory in Debrecen worked out a new method for the well-known colloid reaction based on a quite different base: the China ink reaction of the spinal fluid. This technique is also useful for the examination of the spinal fluid in epilepties, as I will briefly show. The method is very simple and for that reason very useful in general practice. In three small test tubes we put 0·1 c.c. spinal fluid per tube and thereafter in the first one drop, in the second two drops and in the third three drops of ½ per cent. oxalic acid. We add to each tube 1 c.c. of a 1 per cent. solution of the original solution of China ink (Günther Wagner's original 'Perltusch'). We prepare freshly this 1 per cent. solution by diluting the China ink with distilled water.

The dilution of the 1 per cent. China ink solution demands great attention, as the original solution adheres to the pipette and so an additional drop in the contents of the pipette might pass unnoticed. The required solution is prepared with China ink in small flasks and then the pipette is thoroughly rinsed with it.

The result of the reaction can be read after 15 to 20 minutes and also after centrifuging after for a half to one minute. We notice in some test tubes the China ink is completely precipitated and above the precipitate the solution is as clear as water. In other tubes there is no alteration to be noticed; or, in

the intermediary state of the colloid protection to the precipitation, some precipitate is found at the bottom of the tube, over which a pale black solution is visible.

Since the technique of the test and the reading of its results are quite different from those of other colloid reactions, it is not possible to record the results according to the degree of precipitation. We record the result of the China ink test in a new fashion. In the tubes which remain unchanged all the colloid is protected against a precipitating effect, and this we indicate by the sign ∩. When complete precipitation has occurred we put the sign ∪. The intermediary state between colloid protection and precipitation is indicated by —. We write near each other the result read in each tube. The result consequently is in normal spinal fluid as follows : ∪∪∩.

It seems to us, however, practicable to tabulate the result of this test in figures as well. Whereas in normal spinal fluid the precipitates are observed in two test tubes, on the contrary in positive spinal fluid one notes colloid protection ; and for that reason we express the results by the ' colloid protecting index ' (C.P.I.). We obtained the C.P.I. as follows. In the first tube we mark the precipitation as —1, the colloid protection as +1. In the same way, in the second and third tubes are marked —2 and —3 (precipitation), and also +2 and +3 (protection). The intermediary reaction degree in each tube is noted with 0. Numbers so obtained are written in succession, and making a total—we get a series of figures which is called the ' colloid protecting index.' According to this the result is a negative spinal fluid is noted as follows : —1, —2, +3=0. In normal spinal fluid the C.P.I.=0.

In the laboratory of the neurological Clinic in Debrecen we have investigated this test in various diseases of the central nervous system. In cases of epilepsy, as may be seen from the Table, the C.P.I. deviates from the normal in a characteristic way. We usually obtained the following result : ∩∪∩= 1, —2, +3. This means that in epileptics we get mostly the result C.P.I.=2. In general we can say that the China ink test is positive in 87 per cent. of cases of epilepsy, which makes it decidedly superior to the other tests which have been described up to the present.

THE EXAMINATION OF SPINAL FLUIDS OF EPILEPTICS WITH DIFFERENT TESTS, AND COMPARISON OF THE RESULTS. (RESULTS IN PERCENTAGES.)

	Pleocytosis.							
Number of cases	Number of cells 0 in c.c.	Number of cells $\frac{1}{3}$–1 in c.c.	Number of cells 1–2 in c.c.	Number of cells 0–2 in c.c.	Number of cells 3–5 in c.c. slight pleocytosis	Number of cells 5-20 in c.c.	Number of cells 20–50 in c.c. strong pleocytosis	Number of cells more than 50 in c.c. very strong pleocytosis
286	114 39·9	100 34·9	32 11·2	246 86·0	21 (+6) 7·3(+2·1)	6 (+2) 2·1(+1·0)	0(+2) 0(0·8)	0(+2) 0(0·8)

GLOBULIN REACTIONS.
(Pandy, Nonne-Apelt, Ross-Jones, Weichbrodt tests.)

Number of cases	All globulin tests are negative	Pandy + or Weichbrodt ± the others negative	All cases which can be taken as negative	Slight positivity of Pandy test; one or more globulin reactions +	Medium positivity of Pandy and ++ of one or more globulin reactions	Strong positivity of all globulin reactions
325	261 80·5	16 (+15) 4·9 (+4·6)	292 89·80	19 (+10) 5·9 (3·1)	0 (+2) 0 (0·6)	0 (+2) 0 (0·6)

GOLDSOL TEST.

Number of cases	Negative curve	Curve with a slight rise	Medium alteration above the third reaction degree	Curve of meningitic type
300	287 95·6	6 (+5) 2·0 (+1·8)	0 (+1) 0 (0·3)	0 (+1) 0 (0·3)

BICOLOURED MASTIX TEST.

Number of cases	Negative curve	Slight alteration without rise of curve	Slight rise of curve	Medium positivity above the third reaction degree	Curve of meningitic type
289	180 62·2	31 10·7	44 (+16) 15·2 (+5·6)	4 (+10) 1·4 (3·5)	0 (4) 0 (1·4)

SHELLAC TEST.

Number of cases	Negative results	± results	+ results	++ or +++ results
176	146 82·9	7 (+3) 3·9 (1·7)	4 (+7) 2·4 (3·9)	0 (+9) 0 (5·2)

CHINA INK TEST. COLLOID PROTECTING INDEX C.P.I.

C.P.I.=0	C.P.I.=2	C.P.I.=4	C.P.I.=6	China ink test positive	China ink test negative
12·9	77·4	9·7	0	87·1	12·9

Wassermann reaction in spinal fluid negative in all cases (332). Wassermann reaction positive in the blood in 45 cases (13·6 per cent.) out of 332.

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A CLINICAL AND PATHOLOGICAL STUDY OF THREE CASES OF EPIDEMIC ENCEPHALITIS.

By

ARTHUR POOL, Rainhill.

Thesis. "That the explanation of the main features of the symptom-complex known as postencephalitic Parkinsonism is the removal of cerebral inhibition controlling the prespinal sympathetic reflex arc."

It is now about ten years since von Economo gave an accurate clinical description of the disease which has come to be known as epidemic encephalitis. As far as the historical record of disease was concerned, this malady was something new, mysterious in its onset, and appalling in its devastation. Its etiology was a matter of conjecture, its pathology merely guessed at and its sequelæ undreamt of. During the intervening years there has been much clinical material for concentrated study of the disease and yet we must confess that the etiology is still obscure, the causative agent still unknown, the pathological riddle still unsolved and of necessity therefore treatment is practically non-existent. Harvey has told us that "Nature is nowhere accustomed to display more openly her secret mysteries than in cases where she shews her workings apart from the beaten path; nor is there any better way to advance the proper practice of medicine than to give our minds to the discovery of the laws of nature by the careful investigation of the rarer forms of disease." Osler has also reminded us of "the salutary influence of the dead-house"—the place where our clinical acumen is either confirmed or rudely shattered.

In the following study we have sought to practise these guiding rules of conduct. This investigation constitutes a clinical and pathological study of three cases of epidemic encephalitis in an endeavour to correlate the symptoms, signs and histopathology in order to arrive at a working hypothesis of the distinctive clinical picture and phenomena encountered.

CLINICAL DESCRIPTIONS.

1. The first case is that of a male, C. H., aged 28, who was under observation for two years. There was no history of any acute illness prior to admission but the relatives stated that one year previously the patient had complained of transient diplopia and slight headache, since when he had been a little 'queer.'

The mental and general condition of the patient when first admitted were such as to lead to a tentative immediate diagnosis of dementia præcox, and he was thus labelled for about nine months until unmistakable signs of postencephalitic Parkinsonism made their appearance. Mentally he was dull and stupid, excessively emotional, and frequently noisy. There was inverted sleep rhythm, and on occasion he voiced auditory hallucinations. He was constantly complaining of headache and was occasionally guilty of impulsive action.

His general appearance was untidy, the face being expressionless and mask-like. Dribbling of saliva was pronounced and continuous. The posture was that of generalised flexion with festinant gait and slowing of all voluntary movements. There were tremors of the tongue, lips and eyelids and sustained frequency of the pulse rate. The reflex reactions were as follows: Pupils: equal and reacted normally to light and accommodation. Knee and ankle jerks: brisk and equal. Plantar: ambiguous; ? extensor.

2. The second case is that of a male, M. McC., aged 42, also under observation for a little over two years, in whom no previous history of any acute febrile attack could be obtained.

For about three to six months prior to admission he had become gradually depressed and apathetic; easily fatigued, slow in movement—which fact had led to his loss of employment—and finally becoming an excessive burden on his relatives because of inability to attend to his daily toilet, etc.

On his admission to hospital the mental condition was such as to lead to a diagnosis of melancholia, the more prominent features being excessive apathy and depression, complete lack of external interests and social qualities. He complained of headache, dull and continuous. As in the other case it was not until he had been resident in the hospital for some time that the diagnosis of postencephalitic Parkinsonism was made, based on the appearance of the general posture, facies, slowing of all voluntary movements and the continuous dribbling of saliva. In addition there was a coarse tremor of the tongue and a fine tremor of the eyelids. The reflex reactions were as follows: Pupils: equal: sluggish reaction to light and complete absence of response to accommodation. Knee and ankle jerks: brisk and equal. Plantar: bilateral flexor response.

3. The third case is that of a female, L. L., aged 40, under observation from 1925 to 1929. There was a history of an acute attack of encephalitis in 1924, the main symptoms being inverted sleep rhythm, diplopia and permanent strabismus. Almost immediately following the acute attack the patient became very depressed and attempted suicide by jumping from a bedroom window, this fact determining her certification and admission. She was diagnosed as a case of acute melancholia and during her residence she made two further attempts at suicide—by strangling and precipitation. It was not until she had been in the hospital for two years that the unmistakable signs of Parkinsonism made themselves evident. She rapidly developed the complete clinical picture and in addition presented the rather uncommon symptom of palilalia. If asked 'How are you?' she would reply 'My heart's bad,' 'my heart's bad,' 'my heart's bad' *ad lib.* (as many as fifteen repetitions have been noted), the voice gradually diminishing in intensity.

During her residence in hospital she had fairly frequent seizures, i.e., attacks characterised by sudden onset of coma and clonic movements affecting various parts of the body. In November, 1927 the coma associated with one of these attacks was more prolonged than usual, calling for further investigation which took the form of an estimation of the sugar content of the blood and spinal fluid, together with routine examination of the urine. A definite hyperglycæmia and glycosuria were established, with increased sugar content of the fluid. In one of these attacks the patient died suddenly, in February 1929.

The reflex reactions were as follows:—

Date.	Pupils.	Knee and ankle jerks.	Plantar.
1925	Sluggish to light. Normal to accommodation.	Brisk and equal.	Bilateral flexor.
1928	Inactive to both light and accommodation.	Brisk and equal.	Not elicited.

PATHOLOGICAL FINDINGS.

For purposes of brevity and facility of reading we shall consider the three cases together, merely noting points of difference.

In all three no part of the nervous system examined can be said to be completely normal but whereas the first two cases show the chief lesions in basal ganglia, crura, pons and cerebral cortex, the third shows the main incidence of the disease in basal ganglia, lower medulla, and upper cervical cord.

Cerebral Cortex.—In cases 1 and 2 the feature of note is the marked change in the Betz-cells (Fig. 1). None seen are normal. Most cells are swollen and stain very poorly, though some are irregularly shrunken. Nissl bodies have for the most part completely disintegrated and the nuclei are generally

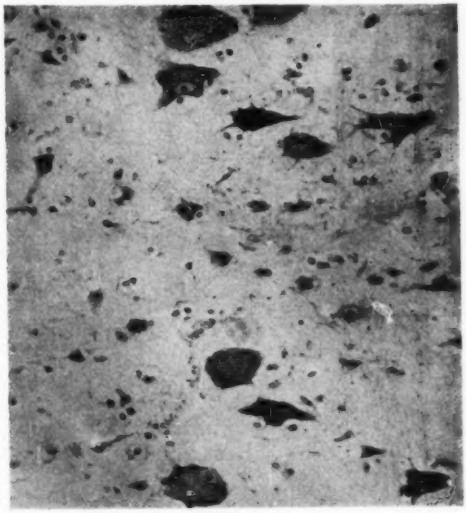


FIG. 1.—Betz cells showing change simulating 'central neuritis'.

excentric. The underlying white matter shows patchy nerve-fibre atrophy. The level of the cortex chiefly affected and the almost complete absence of inflammatory reaction recall the condition to which the term 'central neuritis' has been given (Fig. 2).

Other cortical areas show a similar change in the cells of Meynert and the larger pyramids but of not so severe degree.

In case 3 the Betz-cells are perfectly normal contrasting greatly with the two previous cases. The only demonstrable cortical lesion is found in sections from Broca's area, where within the substance of the white matter

FIG. 2.—Betz-cells from a case of pellagra showing typical 'central neuritis'.

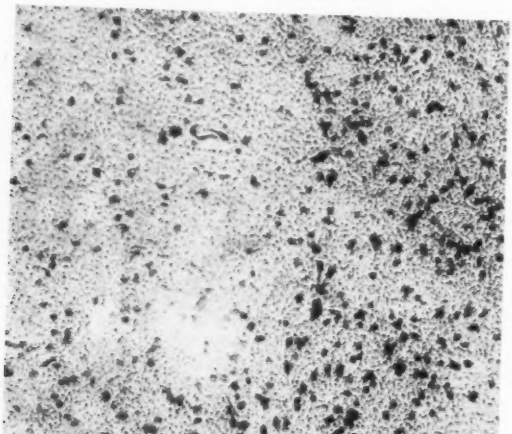


FIG. 3.—Showing one of the subcortical zones of round-celled infiltration in Broca's area of the frontal cortex.

immediately beneath the cortex small zones of round-celled proliferation with accompanying early thickening of the vascular endothelium are encountered (Fig. 3).

Basal Ganglia.—The ependymal lining of the third ventricle and upper part of the aqueduct in cases 1 and 2 shows here and there curious local 'streaks' of proliferating cells penetrating long distances into the subjacent nerve-tissue, and appearing as though they have taken on locally malignant properties. In addition there are numerous subependymal inflammatory streaks. In case 3 the ependyma was completely intact.

Lenticular and Caudate Nuclei.—Cases 1 and 2 show gross degeneration of all types of nerve-cells; the larger are mostly reduced to mere fragments of ragged-looking cytoplasm, while the smaller ones are in an even worse state.

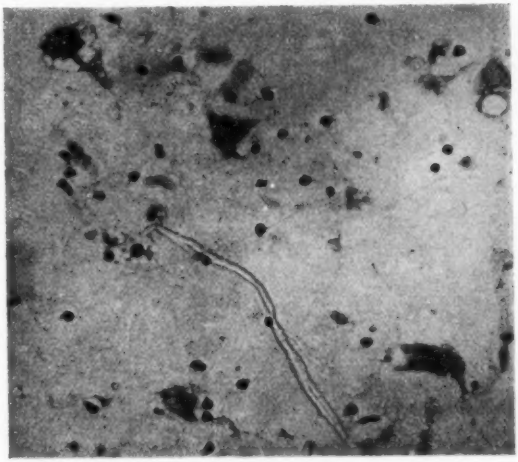


FIG. 4.—Cells from the optic thalamus showing extensive vacuolation.

There is considerable excess of small round cells, perivascular gliosis and endothelial proliferation. In case 3 all neuronic elements are normal but there is definite evidence of vascular upset and round-cell increase.

Optic Thalami.—In the first two cases there is considerable degeneration of many cells and some are vacuolated (Fig. 4), but others are quite normal; i.e., the change here is much less marked than in the adjacent corpora striata. In the third case most cells are normal but a few reveal signs of degeneration. There is pronounced round-celled increase and proliferation of the vascular endothelium.

Crura Cerebri and Hypothalami.—In this area cases 1 and 2 show very severe and extensive damage to the neuronic elements (Fig. 5). Case 3 shows similar changes, but they are less severe and more limited in distribution.

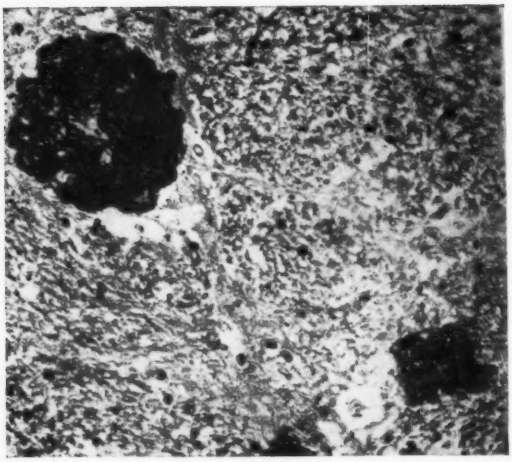


FIG. 5.—Extensive nerve-fibre degeneration in left crus.

In the red nucleus most cells show chronic degeneration, but occasional individual cells appear fairly normal. For the most part the stainable substance of the cells is diffuse and the dendrites appear swollen with ragged fragmented edges. The number of small round cells is extraordinary (often eight, ten or more in satellite relation to one nerve cell) (Fig. 6).

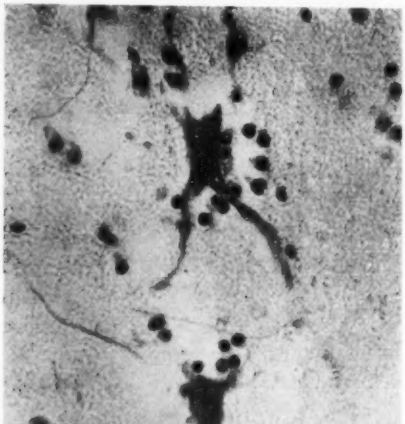


FIG. 6.—Red nucleus. Small round cells in satellite formation to the neurones.

In the substantia nigra the cells are markedly diminished in number, very few cells approaching normality being seen, and even these show chromatolysis and deficiency of processes. Most of the cells seem to have entirely disappeared, their former stations being represented merely by irregular masses of dark pigment. Proliferation of round cells is obvious and the vessels show endothelial changes.

The tegmental areas exhibit similar neuronic, vascular and glial changes but much less pronounced. In all three cases the cells of the third nerve nucleus reveal degeneration of individual neurones. The second case shows the most marked change ; in it we were fortunate enough to have the root of the third nerve, as it emerges from the sulcus oculomotorius, in the section, and very definite atrophy of some of the nerve-fibres was seen.

FIG. 7.—Upper pons. Ependymal proliferation, penetrating subjacent brain tissue.

All the cases show numerous circumscribed areas of nerve-fibre atrophy in the crusta, chiefly located among the pyramidal fibres (Fig. 5).

Pons.—In all cases there is pronounced proliferation of the ependymal cells with local invasion of the brain substance (Fig. 7). There is widespread round-celled infiltration and vascular thickening. In addition, cases 1 and 2 show degeneration of many of the cells of the motor nuclei while others are perfectly normal.

Medulla.—Generally there is excess of small round cells, patchy nerve-fibre atrophy in the region of the pyramidal and rubrospinal groups of fibres and degenerative changes in some of the nerve-cells of the motor nuclei. In addition, case 2 shows widespread degenerative changes among the cells of the olivary nucleus.

Cerebellum.—There is no evidence of any inflammatory upset here, but some of the cells of Purkinje are in various stages of degeneration and in case 2 many have completely disappeared (Fig. 8). The fibres of the superior cerebellar peduncle (connecting the cerebellum with the red nucleus) show patchy demyelinisation.

Spinal Cord.—Cases 1 and 2 merely exhibit very slight atrophy of the fibres in the pyramidal and rubrospinal tracts. Owing to the gradual narrowing of these tracts the atrophy is only apparent in the cervical region of the cord. In case 3 the lower end of the medulla and the upper cervical cord show a most conspicuous lesion, consisting of marked proliferation of the ependyma with surrounding inflammatory exudate and round-celled infiltration.

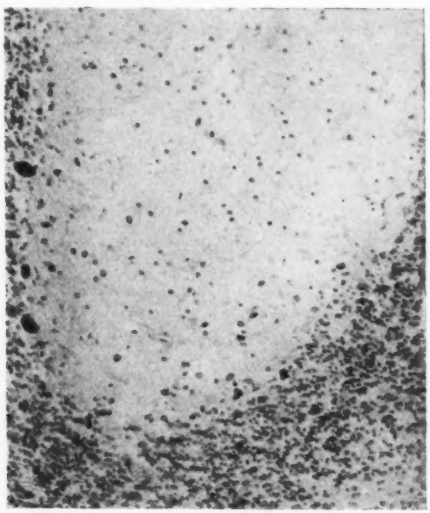


FIG. 8.—Cerebellar cortex. Lower power view of a loop of cerebellar cortex. Normally such a loop would contain about 14-18 Purkinje cells. In this section only 4 Purkinje cells can be recognised. There is no evidence of any inflammation. The lesion is purely degenerative.

In view of the fact that in case 3 clinical investigation revealed a glycosuria and hyperglycæmia it was thought advisable to examine the pancreas. Nothing abnormal was detected.

CORRELATION OF CLINICAL FEATURES AND PATHOLOGICAL FINDINGS.

A consideration of the foregoing pathological findings indicates how widespread is the degenerative and destructive process in the neuraxis involving as it does all parts of the nervous system, from the cortex cerebri above to the

central canal of the spinal cord below, although the main incidence falls on the basal ganglia and hypothalamic regions.

This generalised histopathology releases us from the awkward situation of having to interpret all the symptoms as manifestations of striatal disease alone—a position of precarious tenancy when we reflect on the diversity of the symptoms (hypertonus, tremor, sialorrhœa, glycosuria, palilalia, etc.) encountered.

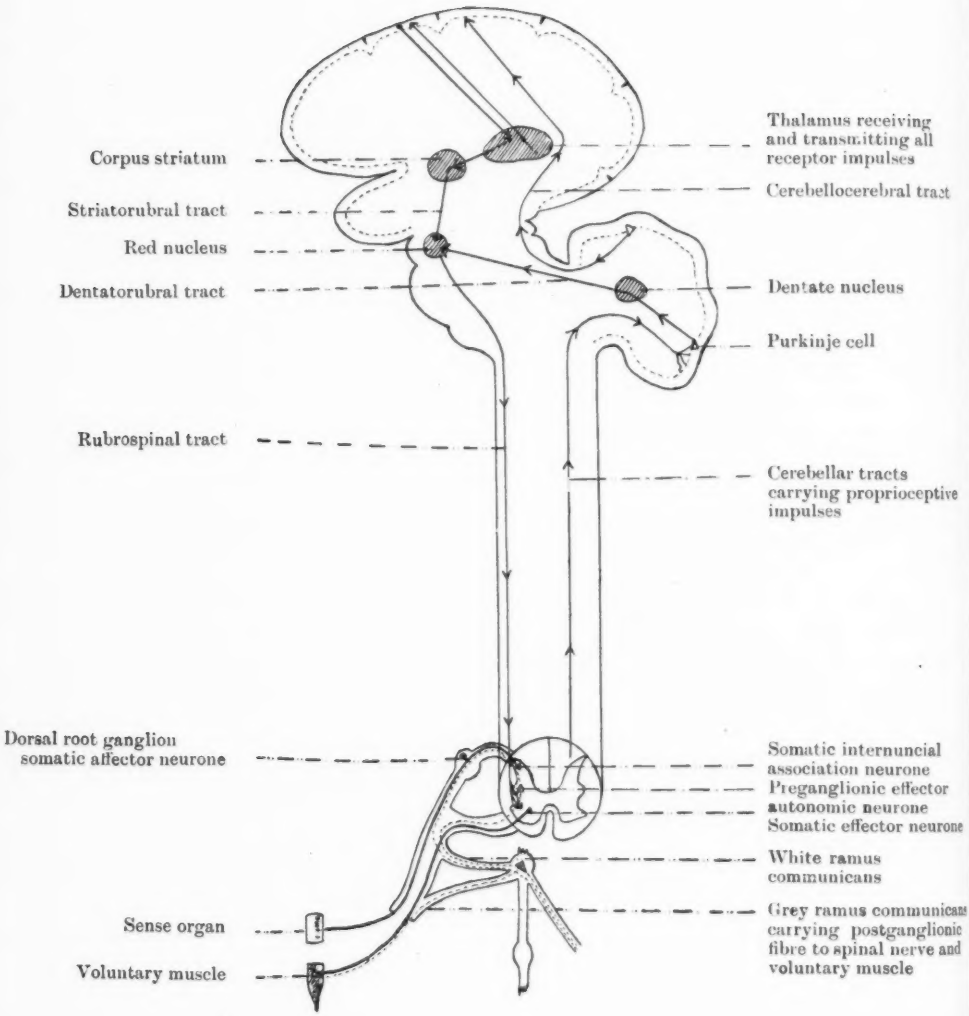
From the photomicrographs it is seen that all the pathological lesions are of a partially or completely destructive nature and it thus follows that the positive symptoms which make up the syndrome of Parkinsonism must be characteristic of release-phenomena, remembering also that the products of degeneration themselves, before they are finally removed by leucocytic activity, may act as irritants to contiguous nervous tissue and thus produce some of the symptoms.

All observers are agreed, and the present investigation confirms, that the main incidence of the pathology centres around the midbrain and basal ganglia and therefore we will deal with this region first. At the outset we find ourselves confronted by a part of the brain in which the anatomical connections are of a most intricate character and even yet by no means fully elucidated. In order to facilitate the further development of our argument we have made a composite diagram of the known connections of tracts and nuclei in this region (Fig. 9). It is purely schematic and is based on diagrams from Quain's *Anatomy* (vol. 3, part I, p. 96) and Berry's *Brain and Mind* (p. 132). The diagrammatic analysis is as follows.

On the affector side, proprioceptive impulses from joint, muscle, and tendon ascend in the spinocerebellar tracts and pass via the peduncles to the cerebellar cortex, whence through the medium of the Purkinje cells and probably other internuncial neurones they are transmitted to the dentate nucleus (from which they are freshly relayed to the red nucleus) and also to the cerebral cortex (either directly or via the optic thalamus). On the effector side we recognise cortical control of the anteroventral thalamic nuclei, which give off neurones passing to the globus pallidus of the corpus striatum, which in turn is connected with the red nucleus. Here this indirect cortical control fuses with cerebellar impulses and thus joint cerebello-striatal control, via the rubrospinal tract, is exercised over the lower somatic and sympathetic reflex arcs.

In the previous section we have noted that the main pathological incidence —of a destructive nature—has fallen on the red nucleus and substantia nigra, with secondary descending degeneration of the fibres of the rubrospinal tract, thus removing the inhibition normally exercised over the lower reflex arcs.

In destructive lesions of the pyramidal system we recognise a distinct clinical picture, the positive signs of which (i.e., everything exclusive of the paralysis) are determined by the release of impulses mediated through the lower somatic reflex arc. Now in destructive lesions of the striorubral system we


Thalamus receiving and transmitting all receptor impulses
Corpus striatum
Cerebellocerebral tract
Striatorubral tract
Red nucleus
Dentatorubral tract
Dentate nucleus
Purkinje cell
Rubrospinal tract
Cerebellar tracts carrying proprioceptive impulses
Dorsal root ganglion somatic affector neurone
Somatic internuncial association neurone
Preganglionic effector autonomic neurone
Somatic effector neurone
White ramus communicans
Sense organ
Grey ramus communicans carrying postganglionic fibre to spinal nerve and voluntary muscle
Voluntary muscle

have a clinical picture quite distinct from that of pyramidal disease and yet the phenomena encountered, as far as the muscular system is concerned, are mediated by the same lower reflex arc (or more correctly, the same final common effector pathway). Seeing that both conditions—pyramidal and Parkinsonian disease—are release-phenomena, it follows that something 'different' or 'extra' is released in one or other of these disease processes. Is there any evidence of this something 'extra' being released? We believe that there is.

The terminal filaments of the rubrospinal tract are known to arborise around the dendrites of the neurones forming the intermediolateral cell column and also around the dorsal cells of the anterior horn. The cells of the intermediolateral column are those which furnish the preganglionic sympathetic fibres, which passing out via the ventral roots of the spinal nerves connect through the white ramus communicans with the sympathetic ganglia (connections and course of the sympathetic fibres are featured in the diagram). From which it is deduced that the something 'extra' which is released by destructive lesions of the rubrospinal tract is this 'sympathetic element.' In support of this contention, i.e., "that sympathetic overaction is explanatory of the main features of Parkinsonism" we submit the following argument.

Hypertonus of a generalised nature (though it affects some muscle groups more than others—thus determining the flexion attitude typical of Parkinsonism) is at once the most outstanding feature requiring elucidation and explanation.

First of all let us remark that a well-established clinical condition in which muscular *hypo*tonus is a pronounced feature—Addison's disease—has been definitely shown to be due to diminution or absence of the secretion of the suprarenal glands, the substance which in an intact nervous system stimulates the sympathetic nerve endings. Again it has been shown that the physical accompaniments of the emotion of fear are determined by transient hyperadrenalinæmia, and anyone who has carefully observed a cat frightened by a dog will have noted the immediate assumption of a generalised attitude of flexion under the influence of sympathetic overaction. Furthermore, Walshe has shown by intramuscular injection of novocain, with resultant temporary abolition of the excess of tone, that the causation of the hypertonus is located in the lower reflex arc; and Royle in a series of experiments on goats claims to have diminished hypertonus by section of the sympathetic—grey rami—fibres to the affected limbs.

This theory of sympathetic overaction as the basis of hypertonus in Parkinsonism has been elaborated by Hunter into a 'dualistic theory' of muscular tonus, and further he has made several dogmatic deductions (e.g., that the sympathetically innervated fibres of voluntary muscle are non-contractile and subserve 'plastic' tone; also "that the posture assumed in Parkinsonian rigidity is due to plastic tone only, exhibited in agonists and antagonists") which have been called in question and shown to be untenable by Kinnier

Wilson, and which have brought the conception of sympathetic overaction into disrepute and called for the numerous denials thereof. While not subscribing to the determined dogmatism of Hunter in support of his dualistic theory of muscular tone, nor on the other hand accepting the denouncement of Adrian (" there is no doubt that the skeletal muscles are connected to the sympathetic system, but the sympathetic fibres are not concerned with tone "), I submit that on anatomical, histopathological, experimental, and clinical grounds there is good reason for suspecting a sympathetic factor in the control of muscular tonus.

Myoclonus and Tremor.—None of the three cases above described showed myoclonus of limb segments, but both the males demonstrated fine tremor of the tongue, lips and eyelids unaccompanied by any wasting of muscles detectable clinically. The tremor became more pronounced when the muscles concerned were put into action and was thus of the ' intention ' variety. The nerve supply of these affected parts is derived from the seventh and twelfth cranial nerves ; and the only pathological counterpart for these clinical phenomena which we could identify was degeneration of some of the cells of the seventh and twelfth nuclei with the presence of much intercellular debris which may conceivably have acted in the role of irritant to the remaining healthy neurones.

Glycosuria and Hyperglycorrhacia.—Much work has been undertaken and a great deal written about the sugar content of the spinal fluid and blood in encephalitis lethargica. Observations by numerous investigators employing seemingly identical methods of estimation have produced diverse results, and while some have claimed that the sugar content of the fluid shows an increase others have just as stoutly denied it. Shrewsbury and Williamson making an extensive study of the subject come to the conclusion that " a *constant* hyperglycorrhacia has not been observed in encephalitis lethargica and the finding of an increased spinal fluid sugar is not believed to be significant of that condition." On the other hand McCowan, Harris and Mann investigated the blood-sugar content of a series of cases of encephalitis lethargica and report thirteen Parkinsonian cases all of which showed a slight hyperglycæmia after the ingestion of 50 gms. of glucose, while out of seven cases of postencephalitis of non-Parkinsonian type only two showed a mild hyperglycæmia. The writer, having had under observation for the past two and a half years some thirty cases of postencephalitis in which sugar investigations have been carried out at varying intervals, can state that some of these cases show persistent hyperglycorrhacia and hyperglycæmia following a carbohydrate meal (i.e., when the liver is full of glycogen) while other cases, more common in the non-Parkinsonian types, show no such upset of sugar metabolism. What is the explanation of these apparently anomalous cases ? All are similar in that they have been infected with the virus of encephalitis, but all do not show upset of sugar metabolism. Therefore the cause cannot reside in the original infection itself but rather in the differing results the infection produces in the nervous

systems of different individuals. In the experience of the writer it is more common to find an increase of the sugar content of the blood and fluid in the Parkinsonian type of case but by no means all of the cases of this type show the change. Is there a feasible explanation of this ? We believe there is.

It will be recalled that one of the pioneers in the elucidation of the problem of glycosuria and hyperglycæmia was Claude Bernard, whose production of experimental glycosuria by puncture of the floor of the fourth ventricle has become a classic of physiology. Subsequent experimenters showed that this effect was only possible when the sympathetic nerve supply to the adrenal glands (splanchnic nerves) and from them the glycogenetic sympathetic nerve-fibres to the liver, were intact and functioning. Langdon Brown, commenting on this experimental glycosuria says, " We know that the main glycosuric effect of Bernard's puncture of the floor of the fourth ventricle is due to the irritation thereby of the splanchnic nerve-supply to the suprarenal glands ". But needle puncture of the nervous system is a destructive lesion, and how can this irritate the splanchnic nerves, which take their origin in the intermediolateral cell column of the fifth to the twelfth dorsal segments of the spinal cord —far removed from the medulla ?

We submit that the explanation is that of a release-phenomenon; destruction of certain fibres of the rubrospinal tract by puncture of the floor of the fourth ventricle releases the corresponding spinal sympathetic fibres from cerebral control, resulting in overaction of the adrenal glands and thus excess-stimulation of the glycogenetic sympathetic nerve-fibres to the liver.

In cases of encephalitis only those which show degeneration of the fibres of the rubrospinal tract will exhibit upset of sugar metabolism (i.e., Parkinsonian cases) and then only those in which the particular fibres, governing the sympathetic nerve-cells connected with adrenal and hepatic control, are affected. We submit that this explanation is tenable on experimental, pathological and clinical grounds above enumerated and also smoothes out the apparent discrepancies in sugar estimations which have been noted by different investigators.

Sialorrhœa.—This is a common symptom in cases of postencephalitic Parkinsonism and occurred in two of those here described. In the experience of the writer, in most cases, though not in all, the passage of time tends to produce a diminution in the degree of salivary dysfunction and cases have been encountered where the symptom has completely cleared up. Can we discover a pathological and anatomical basis for these phenomena ?

The salivary glands derive their dual nerve supply from the cervical sympathetic cord and from the cells of the salivary nucleus (situate in the medulla close to the nucleus of origin of the glossopharyngeal nerve), the fibres passing to the glands via the chorda tympani and branches of the fifth cranial nerve. Physiological experiment shows that the fibres in the chorda tympani are secretomotor and those of the sympathetic are inhibitory.

The two cases in this series which showed this phenomenon disclosed destruction of some of the cells of the medullary nuclei, including the salivary

and glossopharyngeal nuclei, together with a generalised round-celled infiltration. It is conceivable that these products of degeneration act as irritants to the remaining functioning neurones, thus producing hyperactivity—i.e., sialorrhœa. Gradually reparative processes will tend to remove (via the blood stream) the products of degeneration and therefore the irritative factor will slowly be removed with a corresponding gradual improvement of the correlated symptom. That this symptom is an irritative one and not a release-phenomenon is supported by the fact that in the third case showing no sialorrhœa we could find no lesion of the cells of the various medullary nuclei.

Tachycardia.—Sustained frequency of the pulse rate, of moderate degree, has been noted by numerous observers. One of the above three cases showed this symptom. Hall found it in a number of his cases but did not proffer any explanation thereof. Now we know that the heart has a dual nerve supply, the vagus being inhibitory while the sympathetic is both accelerator and augmentor. If we accept the theory that in Parkinsonism the release-phenomena are manifestations of sympathetic overactivity then we have an obvious explanation of the moderàte tachycardia encountered in some of these cases. But why not in all? The cardiac sympathetic fibres come from the cells of the intermediolateral colùmn situate in the upper dorsal cord (segments 1—4). It is a feasible explanation that only those cases in which the corresponding controlling fibres in the rubrospinal tract are out of action will show the sympathetic release-phenomenon of tachycardia.

Palilalia.— The writer has only encountered this symptom in one case, that at present described in this series. It is constituted by a defect (or better a dysfunction) of motor speech. Written speech is apparently unaffected and the sensory side of the speech mechanism is quite normal. The muscular elements concerned in the act of speech (lips, tongue, vocal cords, etc.) have their neural correlates located in the third left frontal convolution, or Broca's area. In our case of palilalia the only lesion of the cerebral cortex we could discover (sections were cut from frontal, central, parietal and occipital areas) was located in Broca's area and consisted of perivascular round-celled infiltration within the substance of the white matter immediately subjacent to the cortex.

Reflex reactions.—In case 1 the plantar response was recorded as ? extensor. In view of the profound degeneration of some of the Betz-cells this observation was very likely correct. In fact considering the case from a pathological standpoint it is remarkable that the pyramidal signs were not more pronounced clinically, but doubtless they were overshadowed by the more definite picture of Parkinsonism.

In case 2 the pupillary reflexes yielded defective response to light and complete absence of response to accommodation. In this case there was pronounced degeneration of the cells of the third nerve nuclei with secondary degeneration of the fibres of the third nerve as it emerged from the sulcus oculomotorius. Similar neuronic changes but of slighter degree were found in case 3, which showed upset of the pupillary responses.

SUMMARY.

The clinical history and histopathology of three cases of Parkinsonian encephalitis are recorded.

The corresponding clinical features and symptoms are related to their possible pathological basis.

It is suggested that the only adequate explanation of the outstanding symptoms which constitute Parkinsonism is that of sympathetic decontrol due to interference with fibres of the rubrospinal tracts.

The symptoms of palilalia and sialorrhœa are probably the result of irritative phenomena due to degenerative products.

Finally, the type of inflammatory cell encountered suggests that the disease is a manifestation of a chronic progressive encephalitis rather than the sequel to an acute infection.

I would like to thank Dr. Reeve for his advice and criticism, Dr. A. G. Clubbe for preparing the diagram and Dr. H. Bean, Chief Laboratory Assistant, for the photography.

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Editorial.

THE DEMENTIA PRAECOX PROBLEM.

SOME years ago, in a preface to the translation of a monograph by Kraepelin, Professor George Robertson observed that by far the most important problem facing the psychiatrist and the community, in the domain of mental hygiene, was that of dementia præcox. From such a view but few psychiatrists would be inclined to dissent—and certainly not those whose lives are spent in daily contact with large numbers of psychotic patients, the majority of whom must be diagnosed as suffering from this sinister disease in one or another of its protean forms. The increasing financial burden imposed upon the community for the treatment of the chronic insane resolves itself very largely into the outlays needed for the care of the almost hopeless victims of dementia præcox. Moreover, as these patients may live to a considerable age under the protected conditions of mental hospitals, they tend to accumulate, and thus form the main reason for the periodical necessity of enlarging existing institutions, and of erecting new ones. Though it is now generally recognised that certain schizophrenic conditions have not necessarily the malignancy that was formerly attributed to them, it has to be admitted that the cardinal symptoms described by Kræpelin as diagnostic of dementia præcox are, in the main, indicative of a disorder the evolution of which is in the direction of permanent psychic deterioration. Clearly, the importance of attacking the problem of the cause of this disease cannot be too emphatically stressed.

During the last few years original and hopeful lines of investigation have been carried out that would appear to have definitely advanced our knowledge of the pathology of dementia præcox. A new orientation was given to biological investigation by the work of Mott on the sex and other glands. His findings were thoroughly in accord with clinical and psychological studies, for in dementia præcox it would seem that it is not so much the highest and most recently evolved functions such as the intellect that are primarily evolved, as the oldest organic-instinctive and affective tendencies which con-

stitute the ultimate urges to action. Further investigations revealed the fact that the morbid changes were not confined to any particular organ, but that every tissue in the body was affected, a condition which was held to be indicative of deficient oxidation processes. This work was followed up by Golla, who was impressed by the fact that, in spite of indisputable evidence of a generalized anatomical change in the tissues, there was no physical symptomatology which would accord with these lesions apart from the very generally observed depression of basal metabolism. The work of Pighini and others of the Italian school failed to discover any single fact in the metabolism that was directly characteristic of dementia præcox and allied psychoses. It seemed that the biochemical and neurological functions of the psychotic were very much like those of the normal subject, except that there was a fairly general, but by no means universal, depression of activity. With these facts in mind Golla changed the methods of attack. He no longer looked for static biological differences between the psychotic and the normal subject, but concentrated on testing how the organism of the former would behave when it was put to any physiological stress. The results of these researches were very striking, and they pointed to the conclusion that in the psychotic subject there is a disturbance of acid-base equilibrium consequent upon the primary depression of the respiratory centre. The highly suggestive and interesting generalisation was made that the physiological condition of the psychotic closely resembles that existing during sleep in the normal individual. It is instructive to note in connection with these researches and their conclusions that Lœvenhart, Lorenz and Waters have independently come to conclusions as to the pathogenesis of dementia præcox which are strikingly in accord with the above, for in a series of experiments carried out upon confirmed cases of catatonic and stuporose precocious dements it was found that all these cases exhibited a marked reduction in the sensitivity of the respiratory centre to carbon dioxide. The opinion is, indeed, expressed by these investigators that the decreased irritability of the cerebral cells to normal carbon dioxide content may be the crux of the psychotic state in question, since they found that a sufficient increase in the carbon dioxide causes a restoration of what we may regard as relatively normal psychic function. The results obtained by Loevenhart and his co-workers were highly impressive. Respiratory stimulation was brought about by inhalations of

carbon dioxide and oxygen, beginning with 10 per cent. carbon dioxide, the strength of which was increased not more than 5 per cent. each minute until a final concentration of 30 to 40 per cent. was reached. The most favourable and striking reactions occurred in those patients who had been mute and inaccessible for long periods of time. A number of patients carried on conversations; one was markedly facetious and made responses typical of talkative hebephrenic dementia præcox. Some of the patients appeared frightened when cerebral stimulation developed; they looked about apprehensively, but became calm and at ease quite promptly when efforts were made to reassure them. In other words, they seemed to comprehend the situation correctly and following this they made coherent and relevant replies to questions. In one patient who had been mute for more than six years, the efforts at speech for about two minutes resulted in a jumble of unintelligible but speechlike sounds, after which the words became distinct and a successful conversation was held. The cerebral stimulation, as evidenced by free muscular movements, animated features, and ability to carry on a conversation and to comply with requests, lasted from two to twenty-five minutes. As the effects of the stimulation passed off, the retrograde changes began. Gradually the voice became less audible; the responses to questions became halting with long lapses; the facial expression became set; eye movements ceased, and attention could no longer be obtained. The muscular rigidity recurred, and in the course of two or three minutes the patient lapsed into his former state of mutism, negativism, and complete inaccessibility. The writers observe how striking it was to note the completeness of resumption of the former muscular state. The production was faithful to the minutest degree—the same posture, the same facial grimace, and apparently the same mental state.

The investigations we have briefly referred to are very encouraging. They certainly throw light on the pathology of dementia præcox and suggest directions in which biochemical therapy might be usefully applied. They show that material advances in our knowledge of the pathology of this widespread mental disease are possible, provided that existing methods of biological research are utilized to the fullest extent, and new ones are devised. There would appear to be no *a priori* reasons why the pathology of this disease should not be eventually elucidated. Possibly, also, with an increased knowledge of

causation suitable remedies may be discovered which may arrest the course of the disease in its early stages or even cure it when fully established. The fact that apparently hopeless dementia præcox cases are capable of being stimulated to a condition of relative normality for even a short time is suggestive and encouraging, and an attitude of pessimism in regard to this disease would appear to be unjustified. There would seem to be no reason to suppose that its nature, prevention, and cure are more impossible than is the case with other forms of physical disease the pathology of which has not at present been elucidated. It is quite certain that, until we have increased knowledge of dementia præcox, efforts in mental hygiene must necessarily lack precision and direction, for this disease is the fundamental problem of psychiatry. Organised, continuous, and unremitting biological research by those who are qualified to carry it out will alone be effective in discovering the secrets of so widespread a psychosis.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[1] **The relation of the level of transection of the brainstem to the occurrence of decerebrate rigidity in young animals.**—W. F. WINDLE. *Jour. of Comp. Neurol.*, 1929, xlviii, 227.

THE author's conclusions are stated as follows.

When young puppies and kittens were decerebrated by sectioning the midbrain at a level bounded dorsally by the cephalic border of the superior colliculi and ventrally by the cephalic border of the pons, the red nucleus was all, or nearly all, removed. These animals exhibited decerebrate rigidity in the extensor muscles of the fore legs and to a less extent in the hind legs.

If the transection was made to pass from the cephalic border of the superior colliculus to the optic chiasma, the red nucleus and other groups of cells forming the hypothalamic nuclei, nuclei proprii pedunculi, and corpora subthalamica were left intact. Animals transected thus retained the ability to right themselves and to progress in the manner normal to animals of like age.

When the transections passed through the cephalic end of the red nuclei or close to this end of the nuclei, the ability of the animals to right themselves and crawl was impaired.

It is evident that the postural reaction is independent of the red nucleus and that it develops before the rubrospinal tract becomes myelinated.

Successful locomotion in young cats and dogs seems to require the presence of an intact red nucleus and possibily other structures in the hypothalamus.

J. V.

[2] **Perception in the region of the optic disc.**—CARLETON F. SCOFIELD. *Amer. Jour. Psychol.*, 1930, xlii, 213.

IMPINGEMENT upon the optic disc of a circular light-point subtending an angle of $\frac{1}{4}^\circ$ produces sensations of light. The occurrence of these sensations is dependent upon the intensity of the impingeing light and the area of the disc upon which the light falls. The threshold for light-intensity is significantly

lower when the light ray falls upon the edge of the disc than when it falls in the centre. A sudden increase in the intensity of the stimulus-light falling upon the optic disc results, under certain conditions, in an impression of movement. (*a*) This impression of movement is more frequent when the stimulus-light falls near the edge of the disc than when it falls in the centre. (*b*) The direction of the movement perceived corresponds to the position of the light relative to the centre of the blind spot, e.g., movement to the right when the stimulus-light is exposed to the right of the centre of the projected blind spot. (*c*) Movement is seen more often in the anticipated direction as the position of the light-point approaches the edges of the blind spot. There is no clear evidence of practice.

These observations seem to offer confirmatory evidence for the hypothesis that the visual phenomena which occur when the blind spot is stimulated by light are due to affection of sensitive retinal areas as a result of the diffusion of light by the refracting media of the eye.

C. S. R.

[3] **Posture of children during sleep.**—M. Adelia Boynton and Florence L. Goodenough. *Amer. Jour. Psychol.*, 1930, xlii, 270.

Changes of posture occurring among a group of 56 children during a total 278 nap-periods in a nursery school were observed and recorded. The number of naps observed for each child varied from 1 to 16. The total number of postural changes observed was 875, which is an average of one change in 25 min. during an average nap-period of 79 min. These children spent the greater part of their total sleeping time on the right side. The left side was slightly favoured, the abdomen ranked next, the back lowest. The rank-order of the four postures is the same when initial posture rather than total sleeping time is considered. A comparison of the sleeping postures most favoured by the individual children with scores on a comprehensive test of handedness does not bear out Sidis' assertion that the most right-handed individuals tend to sleep on the right side. It is suggested that the slight preference for the right side is the result of factors other than handedness, and that individuals in whom hand-preference is more strongly developed are slightly more likely than others to sleep on the side opposite to the preferred hand. Children whose postural habits during sleep are most uniform tend to fall asleep somewhat more quickly than those whose habits are variable. This suggests that kinæsthetic factors are operative to some extent in the induction of sleep. Changes in the posture of the entire body are more frequent than any single kind of partial change in posture. Movements are least frequent during the early stages of sleep.

C. S. R.

NEUROPATHOLOGY.

[4] **Infection and nervous system** (Infektion und Nervensystem).—H. Pette. *Deut. Zeits. f. Nervenheilk.*, 1929, cx, 221.

This is a very long analysis of the relation of infection to changes in the nervous system, and concerns a number of diseased states the precise nature of which is at present very much debated. The paper does not lend itself to brief abstraction, and should be consulted in the original. Dr. Pette deals first with acute inflammatory affections whose lesions are confined mainly to grey matter. Of these he says that the symptoms come on acutely and soon reach a maximum, the spinal fluid points to specific inflammation, both ectodermal and mesodermal reactions occur, and the condition is one characterised by neurotropism. He next examines acute diseases whose lesions involve mainly the white matter. Of these he remarks their onset in acute fashion (as a rule), often in sequence to acute infections of known type (measles, chickenpox, smallpox, vaccination, typhoid, angina, gastroenteritis, etc.); their symptoms vary, the pyramidal system being frequently involved, and depend to some extent on the age of the patient; fluid changes are seldom awanting, and the most characteristic histological change is demyelinisation. Numerous cases cannot be made to fit into any specific disease-form. The etiology and pathogenesis of these respective groups are then discussed at considerable length.

S. A. K. W.

[5] **Infection and nervous system** (Infektion und Nervensystem).—W. Spielmeyer. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1930, cxxiii, 161.

Professor Spielmeyer's new contribution to a much discussed topic cannot be summarised easily. He enlarges on the principles which should guide the research student, pointing out with the greatest clearness that one and the same infection can produce very different alterations in the central nervous system. Reactive glial hyperplasia in infective conditions is often independent of mesenchymal, leuco- and lymphocytic infiltrations. The glia is an interstitial tissue, and the author combats the view of those who argue otherwise. Anatomy has no means at its disposal to determine the etiological nature of pathological processes. The noxæ producing infection in the nervous system may be, and are, utterly different from each other. No etiological deductions whatever can be drawn from identity in histological pictures. Conditions alleged to be due to filterable viruses cannot be distinguished from those attributed to bacterial agencies by their producing glial responses in the first place, and ordinary mesodermal change secondarily. Relative preservation of axis-cylinders in comparison with myelin degeneration is a banal and completely unspecific histological reaction. The author asks what conclusion can be drawn from the very numerous pathological data which he illustrates in

his paper and answers it thus : the study "shows us what is common to the tissue-pictures of completely different infective processes and at the same time what is variable in processes which are etiologically the same."

The article should be read in extenso.

S. A. K. W.

[6] **Non-suppurative encephalitis in childhood** (Die nichteitrige Encephalitis im Kindesalter).—W. SPIELMEYER. *Monats. f. Kinderheilk.*, 1929, xliv, 195.

PROFESSOR SPIELMEYER has discussed in philosophic fashion the bearing of new data connected with encephalitis of postvaccinial and exanthematous types on the problem of the relation of lesions to disease. He is unconvinced by the view which attributes these to activation of a separate virus by that of the original infection. Believing that the actual changes represent something 'new' in cerebral pathology, he underlines the fact that morphology will not explain etiology, supporting his argument by the occurrence of similar or identical cortical alterations as a sequel to a variety of different factors, all affecting modification of the circulation in local fashion. It is the etiology of the conditions concerned that requires research.

S. A. K. W.

[7] **Pathological changes in the region of the tuber cinereum in idiopathic epilepsy.**—LAWRENCE O. MORGAN and HUGH S. GREGORY. *Amer. Jour. Psychiat.*, 1930, ix, 805.

IT is stated that there is no variation in the pathological findings between the cases which may be classed as idiopathic and those where the epilepsy may appear to have been precipitated by cerebral trauma, infection or emotional shock. These findings are as follows :—

1. Shrinkage of the tuber cinereum and cell loss in the substantia grisea ventriculi tertii, nucleus tuberis lateralis and nucleus tubero-mammillaris were seen in all the cases studied.

2. Cell loss is greatest in the substantia grisea and least noticeable in the nucleus tubero-mammillaris.

3. Some degree of cell degeneration is present in the remaining cells of these groups.

4. No correlation is seen between the nature of the cause of death and the amount of the cell loss.

5. Hyperæmia was an outstanding finding in all but one brain and seemed to be regional in the tuber cinereum or at least was most intense there. In the control cases it was entirely absent.

6. These findings in the brains of epileptics, together with the experimental production of convulsive seizures resembling epilepsy in dogs by placing an irritative and destructive lesion in the tuber cinereum, seem to point to this region of the brain as being definitely related to the etiology of epilepsy.

C. S. R.

[8] **Widespread pressure atrophy of the brain, and its probable relation to the function of the Pacchionian bodies, and the cerebrospinal circulation.** —TEMPLE FAY and N. W. WINKELMAN. *Amer. Jour. Psychiat.*, 1930, ix, 667.

ENCEPHALOGRAPHY has called attention to widespread areas of increased fluid collection and cortical atrophy, confined for the most part to the cerebrospinal fluid circulating pathways, over the cortex. A definite pathology of the Pacchionian bodies has been established. In the light of their present accepted physiology, this pathology may predispose or give rise to impairment of cerebrospinal fluid elimination and consequent accumulations of fluid, producing pressure along the course of its cortical pathways. Gross cortical atrophy of the pressure type probably results from a progressive vicious circle. (*a*) Pressure upon the cortex due to gross lesions, as well as fluid accumulations. (*b*) Mild ischæmic changes probably secondary to decrease in circulation. (*c*) Ganglion-cell degeneration and disappearance associated with a fibroplastic glial increase. (*d*) Shrinkage of the cortex and subcortex consequent on pressure and degeneration. Microscopic sections from areas of the same brain in the zones of atrophy show decrease in the number of ganglion-cells and glial proliferation, as well as atrophic changes in the sub-cortex. These areas are usually found in the frontoparietal portions, whereas the temporo-occipital regions are not involved in this atrophy, and sections show changes within normal limits. A possible hydraulic pressure mechanism responsible for the development of certain types of widespread atrophic degeneration of the brain is presented, and may be responsible for many types of gross changes noted, especially in the mentally defective and chronic epileptic or degenerative cases showing atrophy of unexplained origin.

C. S. R.

[9] **Laminar and local destruction of the cortex in a case of amaurotic idiocy with remarks on the theory of ' pathoclisis '** (Sur la destruction laminaire et areale de l'écorce cérébrale dans un cas d'idiotie amaurotique. Remarques sur la théorie de la pathoclise).—MARTHE VOGT. *L'Encéphale*, 1929, xxiv, 509.

THE case of amaurotic idiocy described was somewhat unusual in the degree to which certain layers and areas of the cortex had suffered from the process of neuronal degeneration. In particular in certain areas the fourth layer of nerve-cells had almost completely disappeared. The author uses this case

as an argument in favour of the theory of ' pathoclisis ' or ' tendency to become diseased ' which was enunciated by C. and O. Vogt in 1922. In fact the paper is chiefly valuable in giving a brief summary of this theory, which in its fully developed form is so complex as to be almost incomprehensible.

The paper is illustrated by large-scale photographs of the cortex which are rarely met with except in papers from the Vogts' laboratory in Berlin. In the present instance the degree of enlargement is not sufficient to demonstrate the types of cell present, and the photographs do little more than indicate the zones of greatest cell destruction.

J. G. G.

[10] **A contribution to the study of syphilis of the basal ganglia** (Contribution à l'étude de la syphilis des noyaux de la base).—C. I. Urechia and S. Mihalesco. *L'Encéphale*, 1929, xxiv, 749.

The case on which this paper is based was one of syphilitic Parkinsonism, beginning at the age of 61, and associated with slight dementia. The cerebrospinal fluid was positive to the four classical tests (globulin, cell-count, colloidal and Wassermann reactions). Antisyphilitic treatment however produced no effect. Post-mortem examination of the brain showed numerous senile plaques in the cortex, and a generalised arteriosclerotic condition, probably syphilitic in nature. Perivascular infiltration was very slight in the cortex and rather better marked in the basal ganglia where there was also loss of many of the larger cells and particularly intense changes in the vessel walls. An unusual feature of the case was the presence of nodules of polymorphonuclear cells in the cortex, in the subjacent white matter, and in the red nucleus of the right side. In spite of the absence of spirochætes in these nodules the authors consider them to have been syphilitic.

This combination of chronic arteriosclerotic with acute inflammatory lesions led the authors to discuss the question of the relationship of cerebral, especially mental, symptoms to the syphilitic lesions found in the cerebrospinal fluid or in the brain. This is a big question and the authors do little more than open it by referring to a series of cases of mental disease in syphilitic subjects with ' positive ' cerebrospinal fluids. Obviously a large quantity of carefully examined post-mortem material would be needed if any certain light were to be thrown on this subject.

J. G. G.

SENSORIMOTOR NEUROLOGY.

[11] **Anatomo-clinical observations on myoclonus in epileptics and on related symptom complexes.**—Morgan B. Hodskins and Paul I. Yakovlev. *Amer. Jour. Psychiat.*, 1930, ix, 827.

From a clinical standpoint cases of myoclonus may be divided in two groups : (1) Myoclono-epileptic cases and (2) myoclonic cases without epilepsy. Those of the first group showed pronounced deterioration and symptoms of extra-

pyramidal type, muscular rigidity, mask-like face, hypersalivation; while those of the second group did not show deterioration, remaining in relatively good mental condition until the end, and did not develop symptoms of extra-pyramidal type. In the majority of the reported cases with or without epilepsy, symptoms of cerebellar type were present, being mostly of dyssynergic character, and often showing characteristic skeletal deformities (kyphoscoliosis, club foot). From the standpoint of localization of the pathological lesion, it was found that in cases of myoclonus without epilepsy the lesion affected the dentate nucleus invariably, and in some cases involved the inferior olives as well, but not constantly. Other parts of the central nervous system, and in particular the cortex and basal ganglia, were not involved. In cases of myoclonus with epilepsy, the lesion showed a predilection for the dentate system, being usually more pronounced in the nucleus dentatus but, at the same time, it was more diffuse and showed a severe involvement of basal ganglia and midbrain. The authors are inclined to infer that the myoclonic component in the myoclono-epileptic cases is due to a cerebello- or dentato-tropic lesion, while the epileptic component (deterioration) has for its pathological basis a lesion affecting subcortical grey matter.

C. S. R.

[12] **The interparoxysmal respiratory rate of epileptics.**—S. M. Weingrow *Jour. Nerv. Ment. Dis.*, 1929, lxx, 373.

The observations of the author which are none too numerous persuade him that the respiratory rate of epileptics is lower than that of normals. This rate is not affected by drugs which are being taken by the patient.

R. G. G.

[13] **A clinico-anatomical study of a case of Foerster's syndrome** (Contribution anatomo-clinique à l'étude du syndrome de Foerster).—G. Marinesco and S. Draganesco. *L'Encéphale*, 1929, xxiv, 685.

The case described corresponds more or less exactly to the syndrome described by Foerster in 1909, of hypotonia in a child when lying down and hypertonia when supported in a vertical position, associated with defects of speech and mentality. Choreiform movements of the face and tongue and restlessness of the limbs are common accompaniments of the syndrome which may be congenital. Foerster found the anatomical basis for this syndrome to be a cerebellar sclerosis, whereas in Vogt's case the lesions were in the frontal lobes. In the present case the symptoms began at the age of four years with a febrile illness thought to be malaria. This was followed by a series of convulsions, after which a condition of diffuse hypertonia with trismus set in. This gave place after ten days to the condition in which the patient was found, which persisted till within a few days of death two years later. He could not speak, but uttered a monotonous cry when hurt. He could not feed himself, but masticated food

put in his mouth. Continuous lateral movements of the head were present on lying down and slow forward and backward swaying of the body on sitting up. When lying down the limbs could be hyperextended and hyperflexed, and appeared completely toneless. He could however stand with very slight steadying support, and in this position the limbs were extremely rigid and remained so if he were lifted by the armpits. Along with this hypertonus were athetotic movements especially of the left arm and slow spastic grimacing movements of the mouth and face. The tendon reflexes were active and the plantars flexor in type.

A post-mortem examination revealed an extreme fatty degeneration of the liver, sclerosis of the putamen and caudate nuclei, areas of degeneration in the cortex with thickening of the overlying meninges and some loss of Purkinje cells in the cerebellum. In addition a demyelination of the pyramidal tracts was found which was much greater in the lumbar than in the cervical region of the cord and was not seen in the brainstem or internal capsule. The authors comment on the points of similarity between this case and some cases of hepato-lenticular degeneration, but confess that the syndrome of Foerster appears in other cases to have a totally different anatomical basis.

J. G. G.

[14] **Tumours of the fourth ventricle, studied from clinical, oto-neuro-ophthalmological and neurosurgical points of view** (Rapport sur les tumeurs du IVe. ventricle au point de vue clinique, oto-neuro-ophtalmologique et neuro-chirurgical).—P. MARTIN. *Jour. de Neurol. et de Psychiat.*, 1930, xxx, 255.

THE earliest symptom in cases of tumour of the fourth ventricle is headache, which is often intense and paroxysmal. The pain radiates to the neck and frequently to the shoulders and arms. A stiffness of the trapezii and other muscles of the neck is characteristic, and the patient often holds the head hyperflexed and frequently tilted. At a later stage there appear symptoms of general hypertension and also focal manifestations. The former differ from the hydrocephalic symptoms with other types of cerebral tumour, only in their intensity, their abrupt onset, and their rapid evolution. Focal symptoms include disorders of equilibrium, reeling gait, retropulsion and an attitude of hyperextension. Cerebellar asynergia may be present, and, less often, intention tremor, hypermetria, and adiadochokinesia. Disorders of speech and of writing are usually absent. Tonic attacks may occur, in which the body assumes an opisthotonic attitude, the upper limbs being hyperextended, pronated and adducted. Trismus, irregularity of the heart-beat and Cheyne-Strokes respiration may be noted. These tonic attacks may be precipitated by emotion, sudden noise, or by alterations in posture. In some instances the tonic crises may be more or less unilateral in appearance, thus recalling a torsion dystonia. As the signs progress the tendon jerks gradually disappear. Rare later symp-

toms include spontaneous tremors and various visceral pains. During the terminal phase, bulbar attacks may occur, characterized by tachycardia with irregularity, alternate flushing and pallor of the face, dyspnœa, polyuria and sweating. Sudden death may result.

M. C.

[15] **The cerebral and spinal complications occurring during pregnancy and the puerperium.**—B. J. Alpers and H. D. Palmer. *Jour. Nerv. Ment. Dis.*, 1929, lxii, 484.

This is a comprehensive and long review of all the nervous and mental complications of pregnancy discribed in the literature. These include almost all the conditions familiar to neurologists and can hardly be abstracted. Several illustrations are given and the article must prove a useful reference for those interested in the subject.

R. G. G.

PROGNOSIS AND TREATMENT.

[16] **The relations of extrapyramidal syndromes to the basal ganglia** (Die Beziehungen der extrapyramidalen Symptomenkomplexe zu den Lage- und Bewegungsreaktionen, zum motorischen Haushalt und zu den Stammganglien)—G. Schaltenbrand. *Deut. Zeits. f. Nervenheilk.*, 1929, cviii, 209.

From a series of clinical investigations and physiological experiments the author reaches the following, among other, conclusions.

1. The neck reflexes of Magnus and de Kleijn have no place in the clinical picture of Parkinsonism.

2. Righting reflexes in choreic patients are of normal type.

3. In choreo-athetotic states with the rigidity of Little's disease righting reflexes are awanting or assume a primitive character. In them, further, neck reflexes are seen, but not in cases of chorea.

4. Posture and movement reactions in extrapyramidal conditions show the same imperfections as voluntary movements in respect of inhibition or de-inhibition, slowing, spread of associated movements, and tonization of certain attitudes.

5. Parkinsonism can be produced in animals by acute bulbocapnin toxicosis, but only in restricted dosage. With larger amounts hyperkinesis and epilepsy appear.

6. The corpus striatum does not itself innervate movements, but as a motor centre works in close relation with the centres for voluntary movement, for posture and movement reactions, and with the motor ' Haushaltszentrum ' (hypothalamus).

S. A. K. W.

[17] **Basal metabolism and gastro-intestinal findings in epileptic patients, with a note on treatment.**—T. K. Davis. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 264.

In a small series of epileptic patients there has been found duodenal stasis or some other duodenal abnormality in approximately 29 per cent. It has been suggested that pituitary disorder occurs but no alteration in the sella turcica has been found. There is a distinct tendency for patients subject to epileptic seizures to have a lower basal metabolism than normal and this tendency would seem to warrant the use of pituitary therapy. This was employed along with diet and luminal and although no cure was obtained, 68 per cent. were definitely improved.

R. G. G.

[18] **The treatment of migraine with paracodin** (Migräne-Behandlung mit Paracodin).—K. Kolle. *Münch. med. Woch.*, 1930, lxxvii, 487.

The author draws attention to the value of paracodin, a preparation of dihydrocodein, in the treatment of migraine. This drug, given in the dosage of two, three or four tablets at the commencement of an attack, has, in the author's hands, been of the utmost efficacy in aborting the symptoms.

M. C.

[19] **The treatment of intermittent claudication with muscle-extract** (Die Behandlung der Claudicatio intermittens mit Muskel-extrakt).—M. S. Schwarzmann. *Münch. med. Woch.*, 1930, lxxvii, 758.

Stimulated by the effects of muscle-extract in the relief of angina pectoris and allied conditions, the author has treated a series of patients with intermittent claudication by similar methods. In one patient, the blood pressure fell from 165 to 130, and the dorsalis pedis pulse, which was previously impalpable, was once more felt. Simultaneously, there was a marked subjective improvement and greater power of walking. The patients received up to 26 injections of muscle extract, but it must be emphasized that, in some cases at least, this particular form of therapy was accompanied by dietetic adjustments and reduction of the tobacco.

M. C.

Psychopathology.

PSYCHOLOGY.

[20] **Normal and morbid activity.**—E. Bleuler. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 361.

In this somewhat discursive paper the author lays stress on the role played by instinct in man's activities even in the spheres of ethic and morality. Thus he points out that all activity is ambivalent, there always being an instinctive

inhibition to any impulse. This applies also to sexual impulses, and inhibitions are imposed not primarily by civilization but by instinct. He draws attention to the absence of affect in the encephalitic, in which he differs from the schizophrenic who may not appear to have affective reactions, but who on analysis may display the most intense affect. He points out that the behaviour of the schizophrenic can only be understood on the theory of failures of integration of impulses, so that contradictory affects and impulses may exist side by side without the slightest modification of one by the other.

The paper touches on manifold aspects of psychology and is provocative of much thought, and it is specially valuable in laying stress on the desirability of a biological approach to psychological problems.

R. G. G.

[21] **Children's thinking.**—Victoria Hazlitt. *Brit. Jour. Psychol.*, 1930, xx, 354.

It has been stated that there is no evidence of any specific process of thought that could not be performed by a child of seven. It is here suggested that there is no age limit in relation to the process of thinking, beyond that imposed by lack of experience. If childish egocentrism makes certain processes of thought impossible before seven years of age, how can we account for the evidence of these processes at three and earlier? The truth seems to be that the child's egocentrism is largely due to his lack of experience, which makes him unable to see relations, and his inability to see these makes him egocentric. The assumption that children are naturally non-social in their thinking is being questioned. A further argument against the view that there is any radical difference between the thought processes of the child and of the adult comes from the fact that when the adult has to think about wholly unfamiliar material he makes the same mistakes as the child. He even does it with familiar material if he has not been obliged to concern himself with it previously. Piaget's picture of a striking difference between adult and childish thinking is, the author thinks, due to an over-valuation of verbal expression as a measure of thinking, and an exaggerated view of the logicality of adult thought.

C. S. R.

[22] **Brain and psyche.**—M. Reichardt. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 390.

The importance of this translated article is in its insistence on the importance of the brainstem both in psychology and psychiatry. In it lie the centres of vital function 'where the animal lives' and from it proceed the urges and striving which are integrated and discriminated by the cortex. It is only by an understanding of both parts of the nervous system that psychic function can be understood.

R. G. G.

[23] **Comparative suggestibility in the trance and walking states.**—Clark L. Hull and Betty Huse. *Amer. Jour. Psychol.*, 1930, xlii, 279.

In the present study it was found that waking suggestion required about two and a half times as much time as trance suggestion. If a second suggestion is begun within two minutes after the termination of the reaction to a previous suggestion, and in the same state, the suggestion time of the second is reduced about one fourth. The waking and the trance states do not appear to differ in this respect. The influence of the trance, possibily combined with the additional suggestions given the subject while in it, appears to persist rather strongly after waking. Waking suggestions given a quarter of an hour after waking from a trance require only about 60 per cent. as much time to evoke maximal reactions as when not preceded by a trance. Response to suggestion shows a marked practice effect from period to period, the mean suggestion time being only a little over half for the first period. It has long been observed as one of the most conspicuous characteristics of successive trance inductions that, as a rule, every time a subject goes into the trance the process becomes more rapid.

C. S. R.

[24] **The metabolic rate in emotional moods induced by suggestion in hypnosis.**—J. C. Whitehorn, Helge Lundholm, and G. E. Gardner. *Amer. Jour. Psychiat.*, 1930, ix, 661.

From experiments made it was found that the mood of anxiety or apprehension, hypnotically induced, can increase the metabolic rate. Moods of depression, or elation and of irritability, induced under similar conditions, did not produce any certain increase in this rate.

C. S. R.

[25] **The effects of alcohol and caffeine on intelligent and associative performance.**—Raymond B. Cattell. *Brit. Jour. Med. Psychol.*, 1930, x, 20.

Experiments show that a dose of 20 gm. of alcohol lowers the intelligence quotient and diminishes the power of recalling past experience. Similarly a 0·4 gm. quantity of caffeine is shown to be detrimental to intelligent or associative performance, but particularly to the latter. With amounts of alcohol and caffeine one-half as large as these the general effect tends to be masked by individual differences, but there is some evidence that intelligence and memory are affected oppositely, the former improving on 10 gm. of alcohol and deteriorating under 0·2 gm. of caffeine and the latter showing the reverse behaviour. This, if confirmed, may be of some importance for theories as to the physiological concomitants of the functioning of intelligence and memory. There is little evidence of any dissimilarity of response among the various forms of

recall ; associative reproduction of ideas is impaired under alcohol in proportion to the amount taken and is distinctly improved under the 0·2 gm. dose of caffeine, but the effects of recent associations are more powerful than on old ones. Sex differences of response are not very clearly marked, but there are some indications, the chief of which is that men are more susceptible to caffeine and less to alcohol. On the other hand, some clear age differences are shown, in that middle-aged subjects, in contrast to the majority of the subjects who were in the early twenties, improved in intelligence performance on either quantity of alcohol or caffeine. Compared to the 20-year group the very young group, averaging 17½ years, showed a lesser susceptibility to alcohol and a greater susceptibility to caffeine impairment.

Finally, there are certain well-marked individual differences which manifest themselves most in regard to the response made to the smaller quantities of the two drugs. These individual peculiarities of reaction are revealed to be largely constitutional, in virtue of the fact that they prove to be very similar in blood-relatives. In so far as they correlate with the psychological 'make-up' of individuals they appear to be connected with the particular 'temper' of the conative impulses and to a lesser extent with temperament and character.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[26] **The somatic basis of the neurosis.**—P. SCHILDER. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 502.

A PLEA to regard the neurosis from every point of view, recognizing how the lower centres of the medulla and diencephalon influence the cortex, and the cortex influences the lower centres. Childish experiences are revived by an immediate adult situation and the total picture of behaviour cannot be analysed except by taking all such factors into consideration. In this way we get a dynamic and not a static conception of the neurotic, and a positive idea of his behaviour rather than a negative idea of how he fails to reach normality.

R. G. G.

[27] **The rôle of trauma in various neuro-psychiatric conditions.**—MICHAEL OSNATO. *Amer. Jour. Psychiat.*, 1930, ix, 644.

THE term 'traumatic neurosis' should be limited to cases exhibiting primitive instinctive emotional reactions to injury. These reactions may be considered as being the response of even a normal personality to fear and pain and the other acute stressful features associated with the accident. Thus the ego is overwhelmed and emotional symptoms occur. The term 'traumatic hysteria' should be limited to those compromise-conversion reactions of maladjusted individuals with physical disorders following injury. The 'post-concussion' neuroses are considered as a traumatic encephalopathy. A head trauma may,

by increasing the permeability of the cerebral vessels to the spirochæte and the luetic virus, cause a latent syphilis to become active and cerebrally localized. Psychoses exclusively the result of cranio-cerebral injury are quite rare. The concept ' traumatic insanity ' is a definite clinical entity but is very infrequently seen even with severe head injuries. Trauma, however, even when not directly involving the head, may precipitate a depressive or manic attack in one possessed of the manic-depressive constitution. Trauma alone cannot cause dementia præcox. When it plays a part it acts simply as one of the contributing emotional factors and its influence on the development of the psychosis is fleeting. It may give a temporary emotional colouring to the picture and may supply material ready to hand for delusional elaboration.

C. S. R.

PSYCHOSES.

[28] **The pathological wandering of persecuted individuals** (Note sur les voyages pathologiques des persecutés).—H. Hoven. *Jour. de Neurol. et de Psychiat.*, 1930, xxx, 177.

Individuals afflicted with chronic persecutory delusions may react in one of two ways ; they may defend themselves by active measures such as crying, complaining, accusations, blows, homicidal attacks. Alternatively they may react passively by seeking refuge in flight. The author details six personal cases of vagabondage associated with chronic persecutory psychoses. The flights were not in the nature of impulsive acts but were deliberate efforts at escape. A brief account is given of previous descriptions of this symptom, including Foville's ' aliénés migrateurs ' and the ' maladie du juif errant ' as recorded by Meige. In some instances the journey is undertaken by the psychotic individual under the influence of an imperative hallucination, with the object of fulfilling a mission or a duty. Reference is made to the mystical vagabondage and dromomania as exemplified by the case of Régis. Differentiation has to be made from fugues, which are impulsive acts accompanied by automatism and defects of consciousness, and which may occur in a variety of mental disorders.

M. C.

[29] **The relative importance of hereditary factors in constitutional and organic psychoses.**—C. B. Farr, L. A. Schwartz and L. H. Smith. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 36.

A statistical study of family histories in 200 cases of paresis and manic-depressive psychoses. It was found that the psychopathic taint is quite as great in the paresis group as in the manic-depressive group. This suggests that the incidence of the spirochæte determined the nature of the breakdown rather than the breakdown itself.

R. G. G.

[30] **Psychoses in criminals.**—B. KARPMAN. *Jour. Nerv. Ment. Dis.*, 1929 lxii, 534.

THIS study supports the view that criminality is neither a sudden nor sporadic phenomenon, nor yet an expression of inborn traits. A case of exhibitionism is cited which quite clearly demonstrates the influence of environmental factors in shaping man's antisocial reactions. It is shown that the exhibitionism in this case is really a compulsion neurosis. The study is elaborate but can hardly be conclusive for more than the cases under discussion.

R. G. G.

[31] **Recent statistics of alcoholic mental disease.**—HORATIO POLLOCK and FREDERICK W. BROWN. *Mental Hygiene*, 1929, xiii, 591.

THE number of new admissions to New York State Mental Hospitals increased from 1922 to 1925, but decreased slightly from 1925 to 1926. In spite of this latter decrease, however, the number in 1926 exceeds that of 1922. The rate of new admissions per 100,000 of general population shows a slight decrease during these years. The nine States that were 'wet' before prohibition contributed more than 90 per cent. of all cases of alcoholic insanity in 1922, 1925, and 1926. The percentage of alcoholic cases among all new admissions to these hospitals for the years 1922, 1925, and 1926 shows a steady increase. The percentage of alcoholic cases among all new admissions and readmissions in 1926 was less than one-half that of 1910, but slightly greater than that of 1922 or of 1925.

The following characteristics are noted among the cases of alcoholic insanity in the hospitals of 19 States: Alcoholic insanity is primarily a disorder of advanced middle-age, approximately 75 per cent. of the cases entering between the ages of 30 and 55. The degree of illiteracy among alcoholic patients is somewhat higher than among the general population of the entire United States. More than 80 per cent. of alcoholic cases come from an urban, in contrast to a rural, environment. Over 60 per cent. have been in marginal circumstances previous to commitment, and approximately 25 per cent. have been in dependent circumstances. With respect to marital condition, more than one-half of the patients are married and more than one-third single. The number of single males greatly exceeds the single females, while the number of married and widowed females exceeds the number of males so classified.

C. S. R.

PSYCHOPATHOLOGY.

[32] **The serum cholesterol in mental disorders.**—A. GLEN DUNCAN. *Jour. of Ment. Sci.*, 1930, lxxvi, 284.

THIS investigation was carried out on 336 cases of mental disorder of various types. It shows that in the dull, quiet group of dementia præcox cases the serum cholesterol is high, surpassing the normal limit in the majority of cases,

It is also raised in most cases of manic-depressive insanity and involutional melancholia, and still more so in these cases during a remission. In general paralysis it is variable, but much more frequently above than below the normal, and there is no constant change as the result of treatment. It is high in the majority of cases of psychosis associated with gross organic disease, in alcoholic mental disorders, and in most cases of senile psychosis. It is usually within normal limits in imbeciles, except when complicated by epilepsy. A high serum cholesterol value is to be expected in all types of mental disease, but not in imbecility.

The variations which occur with changes in the mental state are equally definite. Patients in a confused condition, or with excessive emotional reaction, have a lower serum cholesterol content than others in the same psychiatric group, unless the confusion is of very long duration, as in those epileptics who are always confused, when there is little difference. This diminished cholesterol is observed in the confusion or mood changes of dementia præcox, manic-depressive insanity, involutional melancholia and alcoholic psychoses, in the occasional confusion of epileptics, and frequently in association with the epileptic fit.

C. S. R.

[33] **The brain-liver weight ratio in insanity.**—G. W. T. H. Fleming. *Jour. Ment. Sci.*, 1930, lxxvi, 265.

From consideration of the brain and liver weights of 157 epileptics in a serise of 1,932 autopsies, it is definitely shown that in neither emaciated nor non-emaciated epileptics of either under or over 50 years does the degree of atrophy of liver and brain differ from that of the group of psychoses in general. The brain-liver weight ratio deviates less from the normal than in other psychoses. In the psychoses in general in the non-emaciated of less than 50 years of age there is a well-marked decrease in the weights of both the liver and brain. The decrease in weight in the two organs runs parallel, so that the brain-liver weight ratio in this group remains at the normal, viz. ·87.

C. S. R.

[34] **The excretion of phenols in mental patients.**—F. H. Stewart. *Jour. of Ment. Sci.*, 1930, lxxvi, 277.

It is concluded that persons in apparently good health may pass considerable quantities of phenol, *p*-hydroxyphenylacetic acid, indican and indolacetic acid. These quantities are, however, greatly exceeded in intestinal obstruction and cancer of the liver. They were also exceeded in three out of the six psychotics studied from this point of view.

C. S. R.

[35] **The specific dynamic action of protein in relation to mental disease.**—K. E. Appel and C. B. Farr. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 43.

The specific dynamic action of proteins which increase the consumption of oxygen and the basal metabolic rate were determined in a mixed group of mental cases. Some of the schizophrenics showed a diminution of the S.D.A. If this observation is confirmed it would indicate that the administration of anterior pituitary or X-ray treatment to stimulate metabolism might be worth while in early schizophrenia.

R. G. G.

[36] **Basal metabolism and emotional states.**—G. W. Henry. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 598.

An attempt to correlate basal metabolic processes with certain emotional states and their common psychomotor accompaniments. Basal metabolic processes are correlated with emotional states regardless of the clinical type of personality disorder. Elated, overactive, and overtalkative states are accompanied by acceleration of basal metabolism. Depressed, underactive and undertalkative states are accompanied by retardation of basal metabolism. Apprehensive, tense, and agitated states usually imply an acceleration of basal metabolism, but this may be counteracted in some cases in which the feeling of depression is intense. Apathetic states are accompanied by a retardation in basal metabolism, which is not however as great as that associated with feelings of depression.

It is probable that some of the so-called normal variations in the metabolic rate are due to the different emotional states presented by the tested individual.

The basal metabolic rate may be altered to a pathological degree with no other apparent factor than an unusually intense emotional state, and therefore the interpretation of the rate in any given case, regardless of the type of illness, requires the consideration of the prevailing emotional state of the individual tested.

R. G. G.

[37] **Traumatization of the libido.**—L. S. London. *Brit. Jour. Med. Psychol.* ix, 1930, 324.

Previously the writer has demonstrated that the libido, if traumatized, liberates the components of the perversions, homosexuality, and narcissism; and that heterosexuality is supplanted. There must be some embryological defect in the development of the libido that makes certain individuals suscept-

ible to traumatic attacks, and we must concede heredity as an etiological factor in schizophrenia. Here the author demonstrates two cases in which he uncovered an incestuous root of the libido, which in its effort to gratify itself tried to fixate on homosexuality, and that these instinctive impulses were restrained by the super-ego, and the patients sought refuge in symptoms. The libido lives solely for the gratification of its desire, and the components that are unconscious are instinctive. In both these cases the libido was traumatized; in case 1, after an ordinary social contact, it was injured in its attempt to fixate on an individual of the opposite sex. In the traumatization that occurred the components of incest, perversions, homosexuality and narcissism were liberated, and these instinctive impulses not being sanctioned by the super-ego, the patient sought refuge in compulsions and fantasies. In case 2, the libido was traumatized in a love affair, liberating incest and homosexuality, which was not sanctioned by the super-ego; the patient developed a delusion that the girl with whom he was in love was a man. The libido in its attempt to fixate on heterosexuality, which is conscious and is sanctioned by the super-ego, develops a conflict with the instinctive impulses which are unconscious. As a result of these repressed forces and conscious forces a compromise takes place and symptoms are formed. The polar tension between these bipolar impulses, viz., the sexuality and the super-ego, makes the psyche of the schizophrenic labile. Psychoanalysts have shown that the homosexuality can be traced to a strong attachment to the mother. Bleuler was inclined to seek the essential factor in the congenital direction of the impulse; it is on the other hand very clear that in cases of fluctuating sexual direction accident may have a deciding influence. Bleuler concludes that for the present the only hypothesis that can be formulated with any probability is that uranism is a biological manifestation. If it is proved that 2 per cent. of the population are burdened by it, then it may be caused by a heredity mechanism similar to the one that to 100 women 106 men are born. This may also be reconciled with the remarkable circumstance that homosexuality does not die out, despite the fact that it hinders procreation to a high degree.

C. S. R.

PROGNOSIS AND TREATMENT.

[38] **Remarks on psychoanalytic therapy.**—Martin W. Peck. *Mental Hygiene*, 1930, xiv, 67.

In the light of the psychoanalytic conception of the problem of neurosis, its special method of therapy becomes a systematic procedure established to accomplish certain definite ends: (1) to dislodge and recondition certain inadequate and handicapping features of the unconscious mental life; (2) to reduce the deep-lying fear and guilt that have hampered and distorted

psychological growth and development ; (3) to arouse a desire for independence and to force a realization of the futility of trying to appease infantile needs in an adult world—a realization that means something more than mere intellectual understanding, and requires emotional insight and conviction ; (4) last and by no means least important, to emancipate the patient from the transference relation to the analyst. This emotional bond has furnished the central agency for accomplishing the desired changes, but carries the danger of simply shifting from an old bondage to a new one, if dependence on the physician is held to as a way of life, rather than used as a means of therapy. The patient, through the influence of the analysis, has been persuaded to give up an emotional anchorage in his childhood past and achieve a substitute one in the analytical situation itself. A second step of even greater significance must be made. Once more he must renounce an undesirable and compromise adjustment, this time in the analysis, and be launched forth to make terms with the real world. In this world of reality, an appropriately chosen patient, his powers strengthened by the aid of the analysis, has the opportunity to make a normal adjustment as a final substitution and release, both from the childhood complications that imprisoned him in neurosis and from the experience of the analysis that has set him free.

C. S. R.

[39] **The cytology of the cerebrospinal fluid in cases of general paralysis treated by malaria** (La cytologie du liquide céphalo-rachidien chez les paralytiques généraux traité par la malaria-thérapie).—H. Claude and A. Dimolesco. *L'Encéphale*, 1929, xxiv, 528.

The method used in studying the cells of the cerebrospinal fluid was the 'supra-vital' method of examining the centrifugalised deposit when mixed with Pappenheim's methyl-green-pyronin stain. The advantage of this method over the examination of fixed films lies in the possibility of assessing the vitality of the cells by their resistance to the stain. Thus it was found that before treatment the cells absorbed the dye slowly, indicating that they had but recently arrived in the cerebrospinal fluid and possessed considerable vitality. At this period also a great variety of cells was found, although the majority were lymphocytes and medium-sized mononuclears. After malaria the cells diminished rapidly in number and in vitality, so that many of them stained at once, the abnormal cell types disappeared, and in fact the cytology became in every way normal, and usually remained so. If however relapses occurred there was also a return of the typical inflammatory cell picture in the cerebrospinal fluid.

J. G. G.

[40] **The effect of malaria treatment on the lesions of general paralysis** (L'influence de la malaria-therapie sur les lésions de la paralysie générale).—J. LHERMITTE. *L'Encéphale*, 1929, xxiv, 549.

THIS paper is a review of the literature on the subject and does not present any fresh observations. The majority of this literature has been published in Germany and in America, and the conclusions arrived at by different workers have been somewhat discordant. Lhermitte finds however a general agreement in the main facts; the exacerbation of the paralytic process or at least the exaggeration of the cellular inflammatory reaction during the initial phases; then the regression of the process in cases which have survived for some time after treatment, and lastly the tendency to the alteration of the histological appearances from the diffuse lesions of general paralysis to more localised and mitigated changes, resembling more or less closely those of tertiary cerebral syphilis.

J. G. G.

[41] **Discharges against advice from a psychiatric hospital with only voluntary admissions.**—F. L. WERTHAM. *Mental Hygiene*, 1929, xiii, 564.

DURING a period of eight years (1918–1926), 2,758 patients were discharged from the Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital, and during that time 193 patients, or 6.54 per cent. of the total discharges, were discharged against medical advice. Among these there was a predominance of depressions (73) and a relatively large number of cases of general paresis. Manic excitement and paranoid psychoses were infrequent. This would indicate that the social environment of a patient does not easily recognize the milder manifestations of depression as morbid. In elated conditions, on the other hand, the environment takes a very serious view of the patient's state. The large number of general paretics is connected with the fact that, due to its characteristic symptomatology, the patient and his environment frequently have little insight into the seriousness of the condition. In 146 of 193 cases the attitude of the family was the main problem of the physician, as compared with 26 cases in which the initiative could be entirely attributed to the patient. In 10 cases the patient was taken away against his wishes. In many cases the reason given by the family had very little to do with the patient's sickness and treatment, except in so far as it caused a separation of the patient from his family. Out of 172 patients whose further career was obtained, 64 had apparently made satisfactory adjustment outside of mental hospitals; 60 were re-admitted to mental hospitals; 8 committed suicide; 6 made homicidal attempts; 8 made suicidal attempts. In some cases the further fate of the patient did not bear out the prognosis made by the physician.

C. S. R.

[42] **Significant transformations in the administration of criminal justice.**—Sheldon Glueck. *Mental Hygiene*, 1930, xiv, 280.

1. Judicial procedure in criminal cases can be greatly improved by recognizing that the guilt-determining function and the sentencing function should be sharply distinguished in personnel and method. In addition to the trial to ascertain guilt or innocence, a special treatment board composed of persons trained in sociology, psychology, psychiatry, and social case-work technique, should be created.

2. A scientifically staffed clearing-house and detention institution should form an integral part of the board's equipment. Cases should not be disposed of until after careful study.

3. A wholly indeterminate sentence, or at least one with a wide span between maximum and minimum length, is indispensable to proper administration of justice.

4. Prisoners under sentence should be reported upon to the sentencing board at stated intervals, and these reports should be taken into consideration in determining changes of treatment.

5. At each penal and correctional institution there should be organized a department of personal relations with a staff of psychiatric social workers.

6. A bureau of research attached to the sentencing board should be charged with experimentation and research into the principles governing the safe release of various types of offenders on probation or parole.

7. Within institutions, more attention needs to be paid to methods of training and psychotherapy of various types of offenders.

8. Every community ought to have a bureau of prisoners' rehabilitation, staffed by trained social workers, with the duty of educating the public and the police to assume a helpful attitude toward former prisoners.

9. Preventive efforts with children of pre-school and early school age should be extended and improved.

C. S. R.

[43] **Parathyroid therapy in schizophrenia.**—K. M. Bowman. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 353.

The relationship of catatonic conditions to calcium metabolism and parathyroid activity has roused considerable interest. The catatonic cases of dementia præcox here reported were studied in detail, but the results were too inconclusive to warrant deductions and the parathormone used did not seem to influence the blood calcium estimates.

R. G. G.

[44] **The psychiatrist in social work.**—F. J. FARNELL. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 397.

AN admirable common sense article on the dangers of prejudice and lack of vision in social work, no doubt specially applicable at present to the United States but soon to be applicable in this country unless we are careful. Every social worker should read this article and take to heart the aphorism, "All new ideas are not good; but neither are all old and accepted ideas always good or even tolerable."

R. G. G.

Reviews and Notices of Books.

Individual Psychology. By ERWIN WEXBERG, M.D. Translated by W. BERAN WOLFE, M.D. London: George Allen and Unwin, Ltd. 1930. Pp. 442. Price 15s.

THE theory and technique of the individual psychology associated with the name of Alfred Adler are here fully and ably described by one of his pupils, Dr. Erwin Wexberg of Vienna. Adler's general evolution of his theory from its basis in the conception of organ inferiority is sketched in considerable detail, and later developments are also examined, showing that the theory consists of much more than a combination of the elements of 'inferiority complex' and 'will to power.' Character and personality are shown to be compounded of the action of diverse factors, among which the family, social and economic states, sex, and education are all allotted due significance. In Adler's view neurosis arises when the individual seeks to attain personal goals which as a member of a community he "dare not admit to himself, and therefore dare not openly pursue." He may be considered mentally healthy, on the other hand, when "the social feeling has appeared in the conduct of the individual and he has developed the ability to 'give out'." From this general conception the corollary follows that "the mental predisposition to be neurotic is present to a greater or less extent in every human being"; whether it proceeds to a neurosis depends on circumstances. Thus the line between neurosis and health cannot be drawn with any regularity or ease, and the suggestion here made is to the effect that the ability of a man to work or 'hold his job' is a useful criterion of mental normality. The same psychological mechanisms which determine the evolution of the neuroses are responsible for the pyschoses, the essential difference residing in the fact that the 'insane' person has "lost his insight into the nature of his illness and has completely failed to make the proper adjustment to his fellow men."

As a compendium of Adlerian doctrine the book is to be recommended, while the simplicity of the language and the smoothness of the translation enhance its readable qualities.

Insomnia: An Outline for the Practitioner. By H. CRICHTON-MILLER, M.A., M.D., Director, Tavistock Square Clinic. London: Edward Arnold. 1930. Pp. 172. Price 10s. 6d. net.

OF the multitude of available treatises dealing with sleep and its disorders that by Dr. Crichton-Miller is the most recent, and certainly one of the most practical. Considerably less attention is allotted to hypersomnic conditions than to those characterised by insomnia, chiefly because he believes the practitioner sees few of the former cases. Be this as it may, importance is not to be estimated by frequency alone; and for any scientific study of sleep disturbances no inherent reason exists why those of a negative kind should be examined at much greater length than their opposites. But there is doubtless a good practical reason; people mind losing their sleep very much, whereas hours or minutes of extra slumber are not calculated to arouse anxiety, at least in the first instance. Again, states of sleep inversion are significant out of proportion to their numbers, but all that Dr. Crichton-Miller tells us is that "in so far as any post-encephalitic symptom is amenable to treatment, these abnormalities of sleep can be similarly dealt with." Some refractoriness as regards therapeutic measures should not, we think, justify this assumption of a kind of therapeutic nihilism.

The author is at his best in the detailed consideration he furnishes of the multitude of methods—medicinal and psychological—for the successful combatting of insomnia. Throughout this section of the book runs a vein of healthy common sense which must find an echo in the mind of all who have had to deal with the patient no less than with his disability. Dr. Crichton-Miller's book should be a vade mecum for the busy practitioner.

A Textbook of Psychiatry: For Students and Practitioners. By D. K. HENDERSON, M.D., and R. D. GILLESPIE, M.D. Second edition. Oxford University Press: Humphrey Milford. 1930. Pp. 526. Price 18s.

WE reviewed favourable the first edition of this textbook on its appearance in 1927 (this JOURNAL, vol. viii, p. 185). It must be gratifying to the authors to find a second edition called for within a comparatively brief period. Some few pages have been added to the length of the book, which has undergone revision and slight modification. In its present form it is assured of continued appreciation.

Differentialdiagnostik in der Psychiatrie. By Dr. HERMANN HAYMANN, Badenweiler. With a contribution on intelligence tests, by Dr. ERICH STERN, Giessen. Bd. III of Praktische Differentialdiagnostik für Ärzte und Studierende, edited by Prof. Dr. GEORG HONIGMANN, Giessen. Dresden and Leipzig: Theodor Steinkopff. 1930. Pp. 261. Price RM. 17.

THIS closely packed volume forms in reality a succinct treatise on psychiatry, seeing that it deals with much more than simple differential diagnosis. An

introduction discusses general symptomatology in an interesting way, examining in turn somatic and mental semiology, the latter at some length. The chief part of the book provides the reader with a modern account of the symptomatic psychoses, dementia paralytica, involution psychoses, schizophrenia, manic-depressive psychosis, paranoia, epilepsy, and a whole series of 'psychopathies' of heterogeneous class (prison psychoses, narcolepsy, hysteria, pseudologia phantastica, and many more). We do not cavil at the author's selection of epilepsy and narcolepsy for inclusion in his book, nor indeed with his attention to neurasthenia, morphinism, and other states of interest to the neurologist; on the contrary, their description by a psychiatrist is highly desirable. But their more strictly neurological aspect is here rather underestimated, and in one or two points rather misunderstood.

Taken as a whole, the book undoubtedly answers its purpose well and offers the student 'good value.'

The Science of Living. By ALFRED ADLER. London: George Allen and Unwin, Ltd. 1930. Pp. 264. Price 8s. 6d.

How to live well is a theme which has exercised the minds of philosophers and sages since the beginning of time, and there is no good reason for supposing that present-day thinkers have come into possession of a key to the science of living inaccessible to their forerunners of other centuries. 'Le bonheur de ce monde' is but a combination of circumstances and of the individual's way of looking at things, and if stress is being laid today on the betterment of that individual by the application of self-knowledge no claim for novelty in this connexion can be countenanced. The reader in these times is bombarded by psychological treatises and booklets all of which repeat in his ears 'do this and ye shall live,' but whether proof is forthcoming of the superlative efficacy of their several techniques as compared with others, older, and at least equally well-tried, is a matter often conveniently ignored. Little is heard at present of any plea for a simple and normal life unless based on principles derived, as it frequently appears, from consideration of cases of neurosis, misery, and disease. Dr. Adler is no exception; in this readable work of his the following sentence occurs; "Normal children, if there be such, do not concern us." This generalisation scarcely requires comment, so self-revelatory is it. Child guidance is declared to provide the clue for the future avoidance of neurosis and all it brings in its train. Happy marriage is considered "the only satisfactory solution for sex troubles." "The true purpose of a school is to build character." We seem to have heard of these views before individual psychology was invented, and it is no doubt gratifying to see them restated.

The Growth of Reason. By F. LORIMER. International Library of Psychology, Philosophy and Scientific method. London: Kegan Paul, Trench, Trubner & Co., Ltd., 1929. Pp. . Price 10s. 6d.

THIS book is a study of the role of verbal activity in the growth of the structure of the human mind. The author describes himself as an experimental realist and at the end of the book discusses the philosophical implications of the standpoint. The main part of the work is concerned with the tracing of the development of mind from pro-verbal integrations met with in animal behaviour to full logical capacity. In this process the use of words is in the author's opinion essential and he traces the way in which the child acquires words and uses them; also how symbolizations are formed and used as short cuts in thinking; and finally how these are organized into concepts which may be used in rational argument.

A full bibliography is appended and the work is one which is bound to have great interest for educationists and psychologists.

R. G. G.

Études Neurologiques. By GEORGES GUILLAIN, Professor in the Faculty of Medicine, Paris. Paris: Masson et Cie. 1929. Pp. 453. Price Fr.70.

IN the third volume of the series of studies from the author's clinic Professor Guillain presents a selection of his recent contributions to neurology. The work is divided into eight parts under the following headings: (1) cerebral tumours; (2) pathology of encephalitis; (3) pathology of the crus, pons, medulla and spinal cord; (4) spinal cord diseases; (5) pathology of the cranial nerves and roots; (6) muscular atrophies; (7) miscellaneous articles and (8) history of neurology. In part 1 three chapters are devoted to cerebral cysticercosis, a condition seldom seen in this country. A subtentorial tumour described in the same section as an angioglioma seems from the histological description to belong rather to the hæmangioblastomas.

For the most part the illustrations are excellent and like its predecessors the volume forms a series of clinical lessons which should prove of great value to the neurologist.

Syphilis Héréditaire du Système Nerveux. By L. BABONNEIX, Physician to the Charité Hospital, Paris. Paris; Masson et Cie. 1930. Pp. 432. Price Fr.60.

THIS new contribution to the study of hereditary neurosyphilis is based largely on the author's wide clinical experience and consists essentially of descriptions of clinical syndromes of almost bewildering variety. Syphilis is responsible for affections of any cranial nerve and any part of the neuraxis and its coverings.

Its symptoms range from intellectual defect to incontinence of urine. So far as this aspect of his subject is concerned, the conspectus is remarkably complete and equally minute. But inherited neurosyphilis raises also other questions which are here dealt with much more sketchily. A series of cases, it is true, exemplify its occurrence in the second generation, and one, possibly, in the third; further, 20 out of 120 cases seem to prove its causation of epilepsy. But we do not find more than incidental references to blood and spinal fluid examination, the relation of somatic to neurosyphilis, the appearance of clinical manifestations in the same form in parent and offspring, or to data which may be taken to aid in solving the problem of neurotropism where syphilis is concerned. In its class, the monograph has many excellencies and is sure of appreciation.

La Sclérose en Plaques Aiguë. By Dr. ANDRÉ COURNAND, Paris. Paris: A. Legrand. 1930. Pp. 150. Price not stated.

THE exact nosological position of disseminated sclerosis is at present forming the subject of various critical investigations, old though the problem is, and often discussed before. Its relation to what is vaguely styled 'acute disseminated encephalo-myelitis' has not as yet been established. Dr. Cournand, working under the ægis of Professor Georges Guillain at the Salpêtrière, comes to the conclusion that "a large number" of cases placed in the latter category belong as a fact to disseminated sclerosis, and he bases his decision on both clinical and histological considerations, as well as on the results of examination of the spinal fluid. With this conclusion many neurologists will be in substantial agreement, nevertheless we should appreciate the value of Dr. Cournand's work more highly still were we not convinced that until etiological questions are answered it is inadvisable to rely so much as he does on clinical and pathological data, alone.

Die Hypophyse. Lectures delivered in the Vienna Society for Internal Medicine by Prof. Dr. A. EISELSBERG, Prof. Dr. O. MARBURG, Prof. Dr. O. HIRSCH, Prof. Dr. J. NOVAK, Doz. Dr. M. SGALITZER, and Doz. Dr. E. A. SPIEGEL. With 34 illustrations. Berlin and Vienna: Urban and Schwarzenberg. 1930. Pp. 66. Price RM 3.50.

A SLENDER brochure on the pituitary, to which a number of the teachers of the Viennese school have contributed, covers with some completeness, if in a very concise way, the field of present knowledge as regards that organ from its several aspects. Dr. Hirsch has operated on 90 pituitary cases by the intranasal route, of which patients only 17 died within the first three years. No fewer than 27 are still living from four to ten years after operation, and in 17 the condition has improved and been maintained. He alludes also to the technique of pituitary puncture, but is not inclined to recommend it. Dr. Sgalitzer has

treated 25 cases by X-ray radiation, claiming good results in two cases of acromegaly (out of five), in six of lipodystrophic type (of ten), and in four with ocular symptoms pronounced (out of ten). Dr. Marburg's contribution is on the clinical side, while Dr. Spiegel's article on anatomy and physiology is the most detailed as it is the longest in the volume.

An Introduction to the Study of the Nervous System. By E. E. HEWER, D.Sc., London, and G. M. SANDES, M.B., B.S., London. London: William Heineman, Ltd. 1929. Pp. 104. Price 21s.

IN this volume the authors have presented a concise guide to the anatomy and physiology of the nervous system. The style is naturally dogmatic; the greater part of the text is occupied with short descriptions of minute anatomy; physiology is very briefly discussed. The book is profusely illustrated by anatomical diagrams, which will no doubt be of considerable value to the student preparing for intermediate examinations. A chapter is added on certain pathological conditions but this is so brief as to be of little real value to the student of clinical neurology. As a guide to undergraduate students it should prove a useful compendium.

The Neuroses. By ISRAEL S. WECHSLER, M.D. London: W. B. Saunders Company, Ltd. 1929. Pp. 125. Price 18s.

THIS volume is an expansion of the author's short contribution on the neuroses in his textbook on Clinical Neurology. After a short survey of the history of psychiatry and the development of psychopathology, chapters follow on mental mechanisms, the etiology of the neuroses, their classification, clinical manifestations, diagnosis, course, prognosis, and treatment. Though there is some brief discussion of the physiological, neurological, endocrinological, and social-biological views of the origin of the neuroses, psychoanalytic doctrines are in the main advanced and the writer shows himself an ardent disciple of Freud, whose teaching he here closely follows. There is no evidence of any special originality in the work, but the material is clearly treated and the book constitutes a readable presentation of the subject according to the aspect of this particular school of psychopathology. Some concise descriptions of clinical cases illustrating various neurotic syndromes enhance its value. In an appendix some useful advice is given on examination and Dr. David Wechsler adds a chapter on general intelligence, mental level and psychometric tests. For the medical student and general practitioner this monograph amply fulfils its purpose.

C. S. R.

Die Regulierung des Blutkreislaufes. By Dr. W. R. Hess, Professor and Director of the Physiological Institute of the University of Zurich. With 21 illustrations, in part coloured. Leipzig; Georg Thieme. 1930. Pp. 162. Price M 12.

The neurologist will find not a little of direct clinical interest in Dr. Hess's elaborate neurophysiological monograph on the regulation of the circulation, even if there is much more whose heuristic value remains to be determined. A minute study of arterial reaction to chemical stimuli is followed by an investigation of the neural factor in circulatory control and of the role played by venous and splenic elements in the same. A chapter on the regulation of cardiac activity is included. Perhaps the chief value of the monograph rests in its critical analysis of the interrelation of the different components in circulatory function (heart, arteries, veins, capillaries, etc.) and of their synergism and antagonism. The nutrition and depression reflexes of the circulation are also examined at some length. Of these, the former helps to further the service of the circulatory apparatus to the organs it supplies and is adequately stimulated by chemical changes in these tissues; the latter serves to inhibit states of tension in the vascular system and is awakened by stimuli of a physical kind. Difficult and uncertain questions connected with the central (cerebral) representation of vascular mechanisms are discussed in a final section.

The monograph possesses an excellent index and a long bibliography.

Les Acrocyanoses. By Fernand Layani. Paris: Masson et Cie. 1930. Pp. 282. Price Fr.32.

Dr. Layani has written a useful monograph on acrocyanosis, which he defines as a permanent cyanosis of the extremities, painless, coupled with a reduction of skin temperature and with trophic and secretory changes. It is associated also with alterations of a more general kind, viz., hypotonia of the sympathetic system, pluriglandular disorder, and cardiovascular hypotrophy. The condition, which is of minor significance and rarely inconveniences the patient, occurs by itself or in the presence of one or other of the 'ecto-sympathoses' (acroparæsthesia, Raynaud's syndrome, erythromelalgia, sclerodermia, etc.).

For pathophysiological basis the author lays stress on its causation through capillary stagnation the result of capillaro-venous hypotonia; the latter in its turn is attributable to involvement of the vasomotor neurones in organic cases, or to vasomotor derangement consequent on pluriglandular defect in those where no organic affection can be found. Good therapeutic service is rendered by strong doses of extracts of ovary, thyroid, pituitary, and suprarenals.

Lipreading. By IRENE R. EWING, Ellis Llwyd Jones lecturer for training teachers of the deaf, University of Manchester. With an introduction by Sir HENRY A. MIERS, F.R.S. Manchester University Press. 1930. Pp. 74. Price 3s. 6d.

A SMALL primer by an expert contains the marrow of the doctrine and art of lipreading. It offers a complete series of lessons progressive in character, commencing with the rudiments of the subject, and is so clearly expressed as to be capable of being understood by all into whose hands it may come. The deaf person is taught to help himself by mirror study and feeling the movements of his own muscles. By careful and above all by patient study along its lines he will in the end largely overcome the " diabolical disadvantages of deafness " graphically described by the talented authoress of this little handbook.

The Case of Miss R. By ALFRED ADLER. Translated by ELEANORE and FRIEDRICH JENSEN, M.D. London : George Allen & Unwin, Ltd. 1929. Pp. 306. Price 12s. 6d.

THIS is a portion of a life story of a young girl told in her own words. Dr. Adler has never seen her and has no more knowledge of her than the facts which she herself gives and which are here available to the reader. In these pages he has endeavoured to draw a picture of the procedure followed in a psychological analysis according to his school of thought. Throughout, as the story is unfolded, he makes comments and gives explanations and interpretations of the material. In this way he clearly expounds his well-known special views on the problems of the neurotic. Without doubt, there is no better method of driving home any special theory than by presenting an illustrative case, but we cannot help realising that other psychological interpretations, and perhaps as convincing, might in many instances be more feasible. In the case of Miss R., though many of her symptoms can be rationally viewed as originating in her excessive greed for superiority and for victory over her environment, the later fully-fledged compulsion neurosis surely involves other and more subtle mechanisms. The book is readable and of considerable interest, but it is difficult for us to conceive that any psychology can be scientifically based upon such a circumscribed field of reaction. Dr. Adler's enthusiasm and dogmatism are alluring and he has made valuable contributions to the pathology of the neuroses. We must, however, bear in mind that there are other angles of approach to the subject which should not be neglected.

La Statique et l'Hypotonie Musculaire. By Dr. MARINO GOPCEVICH. Paris : Jouve et Cie. 1930. Pp. 207. Price not stated.

THE thesis of Dr. Gopcevich, the work for which has been carried out in the clinic of Professor Alajouanine, illustrates well the application of purely clinical methods to the study of physiological problems. A full description is furnished

of those actually employed (some are novel and all are instructive) in the estimation of static muscular hypotonia as it occurs in a number of differing pathological conditions. A distinction is drawn between postural tonus and postural reflexes, and it is held that the former modifies the latter, which require for their demonstration different technical procedures from those for the other. On the other hand, a postural reflex, or reflex of attitude, is nothing else than the result of the co-ordination of tonic muscular phenomena for purposes of equilibration, and as such cannot be wholly independent of muscular tonus itself. The author argues that the centres for postural reflexes must be at a higher level than tonigenic centres.

The Hygiene of Marriage. By ISABEL EMSLIE HUTTON, M.D. London: William Heinemann (Medical Books), Ltd. 1929. Pp. 125. Price 5s.

PSYCHOPATHOLOGISTS are aware, perhaps more than any one else, how much education on this subject is needed. Such knowledge, too, must emanate from an authoritative source. It is not, however, an easy task to present the necessary information in an ideal form. We feel that this writer has fulfilled the purpose and the whole subject is scientifically and lucidly covered in small compass. In order that the book might incorporate the whole field of reproductive life, new chapters on menstruation, the menopause, and sterility, have been added in this second edition. Other chapters, too, have been amplified and brought up to date. We can cordially recommend these pages to those to whom the problems of matrimony are all-important.

C. S. R.

Occasional Notes.

INTERNATIONAL NEUROLOGICAL CONGRESS, BERNE, 1931.

As our readers know, this Congress has been fixed for the first week of September, 1931. The President of the Congress is Professor B. Sachs of New York, and there are eight Vice-Presidents, viz., Prof. R. Bing, Switzerland; Prof. G. Guillain, France; Prof. M. Nonne, Germany; Prof. Ariens Kappers, Holland; Prof. O. Marburg, Austria; Prof. H. Marcus, Sweden; Prof. O. Rossi, Italy; and Prof. Sir Charles Sherrington, England.

For discussion at the morning sessions the following topics and Chairmen have been selected:

1. Diagnostic and Therapeutic Procedures (surgical and otherwise) in Brain Tumours—Prof. Max Nonne.
2. Muscle Tonus; Anatomy, Physiology, and Pathology—Sir Charles Sherrington.
3. Acute Non-suppurative Infections of the Nervous System—Prof. Georges Guillain.
4. The Role of Trauma in the Production of Nervous Symptoms—Prof. Ottorino Rossi.

The English Committee (Chairman, Sir James Purves Stewart; senior secretary, Dr. Kinnier Wilson, 14 Harley Street, W.1; junior secretary, Dr. W. J. Adie, 86 Brook Street, W.1) has charge of the arrangements for the second of these set discussions, and is able to report that the following names have already been secured in connexion therewith, viz., Drs. Rademaker (Leiden), Spiegel (Vienna), Graham Brown (Cardiff), Bremer (Brussels), Pollock (Chicago), Davis (Chicago), Ranson (Chicago), Negro (Turin), Ramsay Hunt (New York), Weizsäcker (Heidelberg), Denny Brown (London), and Kinnier Wilson (London). The list of openers and contributors is not yet complete.

Further, the English Committee wishes it to be widely known that the afternoon sessions on three days will be reserved for topics which do not fall under the above-mentioned headings. They will accordingly welcome contributions from neurologists and others interested in nervous diseases and request those who can thus aid in making the Congress a success to let the Chairman or the Secretaries have the titles of their papers by November 1, 1930, and to submit abstracts of the same by December 1, 1930. It is most important that this general request should be attended to by all those who desire to take part in the proceedings of the Congress.

Messrs. Thomas Cook & Son have been officially appointed transport agents and in their hands are all matters dealing with travel, fares, hotels, etc.